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# The Australian and New Zealand JOURNAL OF SURGERY

Vol. XXI — No. 1

AUGUST, 1951

## RESULTS OF SURGERY IN CONGENITAL DEFORMITIES OF THE HEART.\*

By C. J. OFFICER BROWN AND KENNETH N. MORRIS.

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IN recent years it has been shown that several types of congenital abnormality of the heart and great vessels are amenable to surgical correction. In the Surgical Thoracic Unit of the Alfred Hospital, Melbourne, we have performed 203 operations on patients suffering from such defects. Our experiences in this type of surgery, and the results which we have obtained, form the material upon which this paper is based. The types of deformity dealt with are shown in Table 1.

TABLE 1.  
OPERATION FOR CONGENITAL DEFECTS OF  
HEART AND GREAT VESSELS.

Patent ductus arteriosus	88
Morbus cœruleus	100
Coarctation of the aorta	11
Congenital vascular rings	3
Aneurysm of the right auricle	1
	—
TOTAL	203
	—

### PATENT DUCTUS ARTERIOSUS.

Closure of the patent ductus arteriosus was the first operation attempted in this series and, in the early cases, this was achieved by two heavy silk ligatures and a wrapping of cellophane. Since 1946 we have used, as a routine, the technique described by Blalock (1946) but we omit the final tie of umbilical tape because we feel

that the presence of a large knot between the aorta and the pulmonary artery might lead to ulceration of the two vessels with recurrence of a shunt. We rely on two purse-strings and one or two running mattress sutures between them, using No. 3 Mersilk for these sutures.

Our experiences with this condition are summarized in Table 2.

TABLE 2.  
PATENT DUCTUS ARTERIOSUS.

- 88 Operations with 1 death.
- 6 Patients did not have a patent ductus arteriosus.
- 77 Ductuses were occluded.
  - Case 1 recurred.
  - Case 2 died from haemorrhage.
- 5 Ductuses were divided and sutured.
  - 6 of these 82 patients had other congenital cardiac defects and 2 of these were not improved.
  - 2 had subacute bacterial endocarditis.
  - 5 had gross cardiac enlargement.
  - 1 had complete heart block and auricular fibrillation with cardiac failure.

The first case recurred because the heavy ligatures were deliberately left slightly loose and it was expected that fibrosis resulting from a wrapping of cellophane would complete the occlusion. The murmur recurred shortly after operation and has persisted.

\*Read at the Annual General Meeting, Sydney, June, 1951.

The second case died because the ductus was torn posteriorly and we did not know how to control the bleeding. Since then sudden haemorrhage has resulted from the tearing of the duct in two cases. In one the tear resulted from a needle puncture which penetrated the aortic wall when placing the purse-string. This puncture was the origin of a rapidly increasing split. The haemorrhage was controlled by the left forefinger while the aorta was freed with the right hand and tapes passed around it above and below the duct. The tapes were tightened until the aorta was occluded and then a forcep was placed on the pulmonary end of the ductus and the ductus was divided. The pulmonary end of the ductus was ligated, and then the aortic end and the adjacent tear were sutured with fine silk. In freeing the aorta the recurrent nerve was damaged and has remained paralysed. In the other case, free haemorrhage occurred after occlusion with two purse-strings and two running mattress sutures had been completed. Forceps were being gently passed behind the ductus to demonstrate it to onlookers when severe haemorrhage occurred. This was controlled with swab and finger pressure, then the aortic arch was dissected free and occluded with tapes, and the ductus was divided. It was found that the purse-string on the aortic side had partially divided the duct posteriorly. This purse-string was removed and the opening in the aorta closed with a continuous stitch of fine silk.

These cases are reported in detail because we believe that if this accident should happen, the only way to handle the situation is to control the aorta and divide the ductus to get access to the site of bleeding. Futile attempts to control bleeding with forceps and extra ligatures are to be deprecated and will usually result in uncontrollable haemorrhage and loss of the patient. Apart from these 2 cases where division became obligatory only 3 other ducts in the series were divided and sutured. In children with small ductuses division and suture is easy and safe, but is unnecessary. In adults where the ductus may be large, tense and friable, we believe that the risk of division and suture will always be higher than that of closure, except in the hands of a very few surgeons with a big experience of this operation.

In one recent case in an adult whose duct was very tense and friable, we partially occluded it with a heavy silk ligature and then placed two finer sutures, carefully drawn tight, on either side of this and finally pulled the heavy silk tight and left it there. The risks of division, or even the insertion of the usual purse-strings, seemed too great in this particular patient.

Some surgeons advise freeing the aorta as a routine before isolating the ductus. We did this once before closing a tense, friable duct.

A few patients have had evidence of some recurrent nerve paralysis after operation, but in all except the one previously mentioned the nerve quickly recovered.

We do not drain the pleural cavity and approximately one patient in ten has required one or two aspirations, but only one has had a significant haemothorax. There have been no empyemata.

We have not completed a detailed follow-up of these patients but all of them have been followed for some months and almost all have been under regular observation. Only one case of recurrence has been seen.

A child with a patent ductus arteriosus may be normally developed and apparently capable of unrestricted activity or it may be weedy and under-developed. After the ductus has been closed the increased well-being shown by even the apparently normally developed child is often striking, and the improvement in under-developed children may be very impressive.

#### COARCTATION OF THE AORTA.

We have operated on 11 patients with coarctation of the aorta. In 9 of the patients, the narrowed segment of the aorta was excised and continuity restored by end-to-end anastomosis, using an everting type of suture, recommended by Gross (1946).

In one of these patients, severe haemorrhage resulted from the tearing of a right intercostal artery and it took nearly two hours to control the haemorrhage before the operation could be continued. A satisfactory anastomosis was completed, but a post-operative haemothorax required aspiration with further transfusion during the night after operation and this patient was in a very collapsed state for twelve hours. Although there was still a considerable amount of clot in the pleural cavity and some fever, his condition caused

no anxiety for the next four days and then he complained of severe pain in his back and became acutely distressed. The clot was evacuated from his chest but he died shortly after this operation. At autopsy the anastomosis was intact but there was a long tenuous clot in the aorta, extending from the anastomosis into the sub-clavian artery and back along the arch through the aortic valve to the mitral valve. It was attached to the anastomosis and to the mitral valve.

It seems probable that this thrombosis developed during the period of post-operative collapse. The haemothorax resulted from bleeding from the wound in the chest wall. Great care is needed in closing the chest in these cases to see that haemostasis is complete. Small amounts of bloody effusion were aspirated from the pleural cavity post-operatively in 3 other patients and to avoid this, a catheter drain has been used in the last 5 patients and removed after twenty-four to forty-eight hours.

Case 3 was mildly febrile and had had a series of small tender nodular spots, suspected of being embolic, in her lower extremities for two months before operation. It was felt that she probably had some endarteritis at the site of her coarctation. Blood cultures were negative but, as we did not take the blood from the arteries of the lower limb, this finding was not significant. In this patient fever persisted for two weeks after operation and she developed a small empyema that required drainage. After this her fever subsided.

In one patient (Case 6) the area of constriction was too long for direct aorta-aorta anastomosis, therefore the subclavian artery was ligated and divided high in the chest and turned down to be anastomosed to the divided aorta below the constriction.

In only 3 patients was it possible to place a clamp on the descending aorta above the constriction. In the other 6 patients who had a direct anastomosis, control of the upper end was obtained in 4 by occluding the aorta between the left common carotid and the left subclavian arteries and by occluding also the left subclavian artery. In the other 2 a Willis Pott's clamp was placed on the arch of the aorta at the level of the origin of the subclavian artery in such a manner that the aorta was occluded without completely obstructing the subclavian artery. In 6 cases there was some leakage from the suture line, controllable by sponge pressure after the clamps were removed. In these cases a strip of gelfoam wrapped around the anastomosis and left in place produced complete control of bleeding. It was felt

that this was better than waiting for final control by sponge pressure, or the placing of additional sutures. The oozing appeared to come from needle punctures and additional sutures only created fresh punctures which required further control.

In the 9th case (a girl of 24) thoracotomy only was done. The aorta was narrowed for a length of  $3\frac{1}{2}$  inches and was considerably calcified. Immediately below the arch there was a firm calcareous nodule and then a narrowing. Below this narrowing there was another firm nodule below which for a length of approximately 2½ inches the aorta was about the diameter of a lead pencil and irregularly calcified. The aorta then widened out to a diameter of about three quarters of an inch, about 2 inches above the diaphragm. Resection in this case was obviously impracticable. Review of the lateral film showed evidence of calcification that had not been suspected before operation.

The changes in blood pressure following operation are shown in Table 3.

In case 6, aortic-subclavian anastomosis was done because it was felt that the coarctation was too long for direct anastomosis. In all others, excision and direct anastomosis was used.

In 5 cases, Nos. 1, 3, 7, 8 and 10, blood pressure readings in the arms and legs are now within normal limits. In 2 cases, Nos. 4 and 5, there has been a satisfactory lowering of blood pressure in the arms but blood pressure in the femoral arteries is still below that in the arms. In case 6, where a subclavian artery anastomosis was done, the blood pressure in the arms has not been greatly reduced and there has been no significant increase in the blood pressure in the femoral arteries. Clagett (1948) used this technique in 8 of his first 21 cases and found that by and large, post-operative results were not as good as it was hoped they would be. The subclavian in this case was very large and the reconstituted aorta was very little smaller than normal but as Clagett (1948) points out, the vascular bed through which the heart empties itself is not increased by joining the subclavian artery to the aorta. Normally the pressure in the femoral arteries is about 20 mm. above that in the arms and a completely satisfactory operation should restore this ratio as well as reduce the brachial pressure.

Case 11 was operated on at 10 years of age because his heart was progressively enlarging. His blood pressure was not dangerously high and femoral pulses were palpable though faint and delayed.

TABLE 3.

Case No.	Initial	Age	Sex	B.P. before Arms	Opn. Legs	B.P. after Arms	Opn. Legs
1	B.Y.	20	F	180/110	Not recordable	150/90	160/90
2	L.W.	22	M	170/95	100/85	Post-operative Death	
3	N.M.	15	F	180/105	Not recordable	135/70	140/90
4	K.F.	15	M	170/80	Not recordable	130/80 150/85	125/95
5	R.B.	19	M	210/103	Not recordable	155/85	110/90
6	A.C.	32	M	220/80	140/90	195/70	115/103
7	B.F.	22	F	195/90	Not recordable	115/65	135/130
8	J.T.	18	F	195/105	Not recordable	150/95	175/135
9	I.C.	24	F	176/110	Not recordable	Inoperable	
10	H.B.	20	M	200/100	Not recordable	170/100	190/110
11	W.G.	10	M	140/80	100/75	100/50	130/70

Blood pressure readings were obtainable in his legs. Seven days after operation his blood pressure in both arms and legs was considerably higher than the pre-operative readings. The ratio between arms and legs was normal. Two weeks after operation his brachial blood pressure had fallen to a satisfactory level and the leg-arm ratio was maintained.

#### CONGENITAL VASCULAR RINGS.

Three examples of this anomaly have been operated on.

Case 1, a girl aged 13 months, had a constant loud wheeze and regurgitated her food. X-ray examination was difficult and no satisfactory films were obtained but a right aortic arch was suggested. The condition present is illustrated in Fig. I. She was treated by division of the obliterated ductus and the aberrant left subclavian artery which rose from the descending aorta behind the oesophagus. Her symptoms were immediately improved but some wheeze persisted. Now, at the age of 3, the wheeze has completely disappeared and she is eating normally and looks very well.

Case 2, a woman of 42, had difficulty in swallowing all her life and lived on semi-solids. The aortic arch was right-sided, crossing posterior to the oesophagus and indenting it markedly. Constriction was produced by a short ligamentum arteriosum tying the pulmonary artery back against the aorta and imprisoning the oesophagus and trachea in the ring thus made. Division of this

band, even at the age of 42, has enabled her to swallow normally. Figs. II, III and IV illustrate the condition.

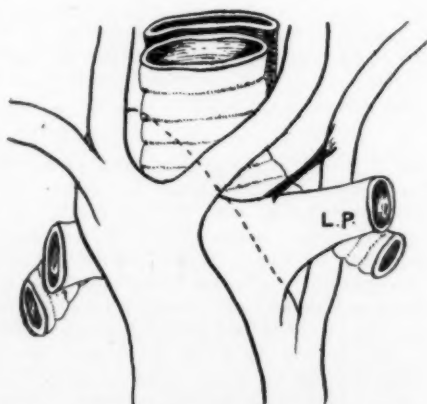


FIG. I. E.G., aged 1. CONGENITAL VASCULAR RING. Right aortic arch and descending aorta crossing gradually to left side with aberrant left subclavian artery rising from the aorta behind the oesophagus. The obliterated ductus passed from the pulmonary artery to the left of the trachea and oesophagus to join the left subclavian artery. The ductus was divided and the left subclavian artery resected from its origin to the level where the ductus joined it. L.P.—Left pulmonary artery.



FIG. II. I.L., aged 42. Postero-anterior film showing right sided aortic knob.

Case 3, a girl of 2, had a constant wheeze and frequent attacks of bronchitis. Barium meal showed some distortion of the oesophagus, and lipiodol in the trachea showed very considerable narrowing of this tube at the level of the pulmonary artery.

Again the aorta was right-sided. At operation a tight ligamentum arteriosum completed a ring formed by the pulmonary artery and the aortic arch. Division of this band produced definite improvement but the result at the time seemed disappointing. Now, at the age of 3½, she has lost all her wheeze and has had no respiratory infection for some months.

Even after the condition has been relieved, symptoms may persist until growth of the air passages completes the cure.

#### ANEURYSM OF THE RIGHT AURICLE.

A boy of 6 presented with what was considered to be an anterior mediastinal tumour, probably a teratoma (see Figs. V and VI). Operation revealed a large deficiency of the pericardium with a very thin-walled right auricle bulging through the defect. Nothing was done and now the heart is steadily enlarging. The boy is still capable of a fair amount of activity (See Fig. VII).

Could the bulging auricle have been amputated and repaired and a graft of fascia lata used to repair the pericardium?



FIG. III. Oblique film shows how the oesophagus is kinked forward over the aorta and pushed back below this by the left pulmonary artery. Same patient as in Fig. II.

#### PULMONARY ATRESIA AND STENOSIS.

Methods of surgical relief for the patient with a patent ductus arteriosus, a coarctation or a congenital vascular ring are

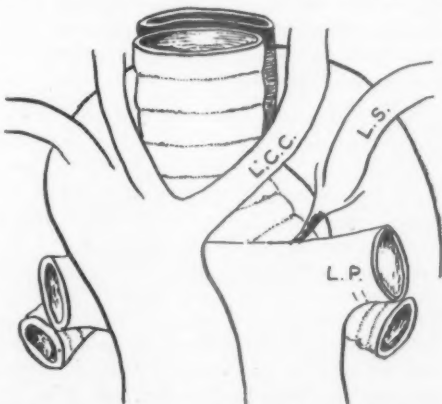


FIG. IV. A right aortic arch crossed immediately to the left side behind the oesophagus; the pulmonary artery was tied back in front of the oesophagus and trachea by a short obliterated ductus passing to the left of these structures. The obliterated ductus was divided and a few fibrous adhesions freed. L.C.C.—Left common carotid artery. L.S.—Left subclavian artery. L.P.—Left pulmonary artery.

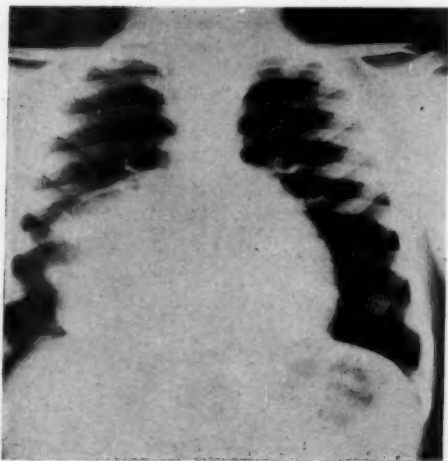


FIG. V. Aneurysm of the right auricle. Postero-anterior film showing the "tumour" to the right side of the heart before operation.

straight-forward anatomical conceptions and were delayed in achievement because of the difficulties and technical problems presented. In the surgical treatment of pulmonary stenosis Blalock and Taussig (1945) introduced an entirely new physiological conception. In operating on a patent ductus arteriosus or a coarctation, the surgeon aims at leaving the patient with a normal heart and great vessels. In Fallot's tetrad it is impossible to make the heart normal, but by adding another defect—an artificial ductus—the surgeons aim at compensating for the original deformity.

We have performed 100 operations for this condition in 95 patients. Five patients were operated on on both sides. In 2 of them there was no adequate pulmonary artery on either side. Two others with right aortic arches were inoperable on the left side because the subclavian artery had no communication with the aorta (See Fig. VIII). It was connected to the pulmonary artery by a fibrous cord representing the ligamentum arteriosum. In both, a satisfactory anastomosis was later done on the right side. In retrospect it was realized that both these patients should have low pressure subclavian systems in the left arm. In both cases radial pulses were absent and blood pressure readings not obtainable. It is suggested that



FIG. VI. Lateral film of the same patient as shown in Fig. V.

the pulse and blood pressure should be carefully checked in both arms before operation in patients with Fallot's tetrad and hypotension in one arm should contra-indicate operation on that side. In one patient, left subclavian pulmonary anastomosis produced

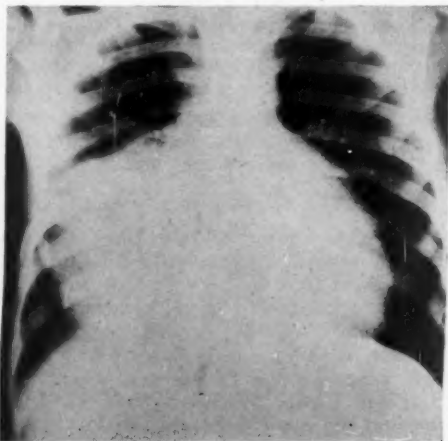


FIG. VII. Postero-anterior film of the chest two years after the film shown in Fig. V. The heart has increased in size.

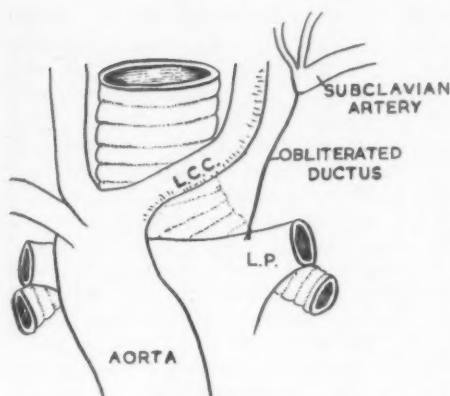


FIG. VIII. FALLOT'S TETRAD, with right aortic arch and left subclavian artery not connected to aorta but joined to pulmonary artery by a long tenacious obliterated ductus. L.C.C.—Left common carotid. L.P.—Left pulmonary artery.

no improvement. It seemed that the anastomosis was not working and the operation was repeated on the right side with complete satisfaction. Fifteen operations in 13 patients were abortive because an anastomosis could not be carried out. Two of these patients already mentioned were relieved by operation on the other side, so that out of 95 patients, 11 proved to be inoperable and thoracotomy only was done. Table 4 summarizes the conditions which prevented anastomosis in these 15 operations.

TABLE 4.

No adequate pulmonary artery	9
Eisenmenger's complex	1
Transposition of great vessels	1
Left pulmonary artery supplying only upper lobe	1
*Left subclavian artery not connected to aorta and too high for anastomosis (R. aorta)	2
Closure of pulmonary artery stopped heart	1
TOTAL	15

\*Both operated on successfully on other side.

Eighty-five anastomoses were completed in 84 patients. At the beginning of this work we planned to use the left pulmonary artery

in all patients if it were available and to do a Willis Potts (1946) pulmonary-aortic anastomosis when the aorta was on the left side, and a Blalock's subclavian-pulmonary anastomosis when the aorta was on the right side. We followed this plan until we had operated on 30 cases but with increasing experience tended to do more and more Blalock's operations with a left-sided aorta. We still prefer to use the left pulmonary artery if possible but in the last 43 cases have used the Willis Potts technique only once. In all cases we have used a posterolateral incision, resecting the 4th rib and we believe that for either operation this gives a better exposure than the anterior incision recommended by Blalock. On the right side particularly, the anterior incision necessitates retraction of the superior vena cava and more interference with the heart than does the posterior incision, and this may be a factor in the almost complete immunity to cardiac disturbance that we have noticed in our operations. The Willis Potts operation is difficult on the right side and we have not used it on this side. Even now, we are not convinced that one operation is better than the other, but we shrink from interfering with the aorta when the same result may be obtained by sacrificing a non-essential vessel like the subclavian. We always isolate the subclavian and its branches as high in the neck as we can reach and we have never seen any disturbance in the function of the hand as a result of this peripheral division of the subclavian and its branches. Several of our patients have had a Horner's syndrome after operation and in some cases this has persisted. We have never used the innominate or the carotid arteries because of the hazard of cerebral complications. The use of these vessels should be considered only in desperate need when no other method is available.

Table 5 summarizes the anastomoses we have done.

Under the age of three diagnosis is difficult. Operation is avoided under this age unless the patient's condition makes it imperative and it is in this group that inadequate pulmonary arteries are most likely to

TABLE 5.

		Deaths
Aorta-pulmonary artery (L) (Willis Potts)	33	4
Left subclavian-pulmonary anastomosis		
End-to-side	40	4
End-to-side with division of pulmonary artery	1	
End-to-end	7	1
Right subclavian-pulmonary anastomosis		
End-to-side	2	
End-to-end	2	
	—	—
TOTAL	85	9
	—	—

be found. The risk of operation is higher and all 3 of the inoperable cases that we explored in this group died. At any age, exploratory thoracotomy carries a higher mortality than successful anastomosis, and out of 15 abortive thoracotomies carried out on 13 patients there have been 5 deaths. This compares very unfavourably with 9 deaths in 85 anastomoses completed in 84 patients. The youngest patient operated on was aged 4½ months and the oldest 27 years. In both of these, satisfactory anastomoses were completed. Table 6 summarizes these results.

TABLE 6.

Age	Anastomosis		Thoracotomy Only	
	Operations	Deaths	Operations	Deaths
3 or less	10	2	3	3
3-13	58	3	11	1
13 or over	17	4	1	1
Totals	85	9	15	5
Patients	84	9	11	5

## CAUSES OF DEATH.

Table 7 shows the causes of 14 operative deaths.

TABLE 7.

- |  |                    |
|--|--------------------|
| 1. Absent left pulmonary artery                                    | } Thoracotomy only |
| 2. Transposition of great vessels                                  |                    |
| 3. Transposition of viscera<br>Inadequate pulmonary artery         |                    |
| 4. Absent left pulmonary artery                                    |                    |
| 5. Absent left pulmonary artery                                    |                    |
| 6. Haemorrhage at operation (P.A.)                                 |                    |
| 7. Haemorrhage at operation—Death 6 weeks later (P.A.)             |                    |
| 8. Haemothorax (S.P.E.S.)  |                    |
| 9. Probably diphtheria—Tracheal obstruction<br>—Tracheotomy (P.A.) |                    |
| 10. Pulmonary oedema (P.A.)  |                    |
| 11. Pulmonary oedema (S.P.E.S.)                                    |                    |
| 12. Tricuspid stenosis — Cerebral damage (S.P.E.E.)                |                    |
| 13. Haemothorax, etc.—Death 6 weeks later (S.P.E.S.)               |                    |
| 14. Cerebral vascular damage (S.P.E.S.)                            |                    |

P.A. = Pulmonary-aortic anastomosis.

S.P.E.S. = Subclavian-pulmonary end-to-side anastomosis.

S.P.E.E. = Subclavian-pulmonary end-to-end anastomosis.

It will be noted that 4 of the 5 deaths in cases where thoracotomy only was done were in patients without adequate pulmonary arteries. They were so severely handicapped that they could not tolerate the operative interference. One of these died as the operation was being concluded and the other three some hours after operation. The fifth patient who died following thoracotomy was a girl of 15 with very gross postural scoliosis which made proper interpretation of X-rays of the heart impossible. Thoracotomy showed that she had transposition of the great vessels and she died four days after operation. Of the 9 operative deaths following anastomosis 4 were directly or indirectly due to haemorrhage — from the anastomosis in 2 and from the chest wall in two. In 2 early cases where the Willis Potts technique was used in young adults, bleeding from the anastomosis seemed excessive and attempts were made to re-apply the clamps and put in extra sutures. In one case this resulted in tearing the anastomosis apart and the boy died as it was being repaired. In the second patient a few extra

sutures stopped the bleeding. He was very collapsed and recovered slowly. Two weeks later he had an embolic occlusion of his right common iliac artery. Embolectomy restored the circulation in his limb but he died six weeks after his anastomosis. Autopsy showed an old ball-thrombus in his left ventricle and the embolus had come from this. The anastomosis had been completely closed by the extra sutures inserted to stop bleeding but there was no clot around it. With a little more experience it would have been recognized that the bleeding from the anastomosis in both these patients would have stopped with the application of swab pressure for a few minutes.

Post-operative haemothorax occurred in 3 patients and 2 of them died. In all of them an X-ray taken twenty-four hours after operation was satisfactory and showed only a small amount of fluid in the pleural cavity. Later in the day, signs of haemorrhage appeared and each patient then was found to have a massive haemothorax. One was recognized too late. Another was aspirated and the patient recovered. The third was aspirated and the patient, being transfused, improved but did not progress satisfactorily and died six weeks after operation with a small empyema and infarcts in the kidneys and spleen. At autopsy in both of the patients who died the anastomosis was satisfactory and we believe that the bleeding in these cases came from the chest wall. All these patients were between 18 and 24 years of age.

Two deaths occurred from cerebral damage recognised shortly after operation. One was a baby of 13 months weighing only twelve pounds with tricuspid stenosis and with tiny pulmonary arteries. An end-to-end subclavian-pulmonary anastomosis was done but the child never recovered consciousness and died some hours later. The other was a girl of seven in whom a satisfactory end-to-side subclavian anastomosis had been done. She remained drowsy and died on the third day. Pulmonary oedema accounted for 2 deaths. A boy of 6 appeared in excellent condition six hours after a satisfactory anastomosis of the left pulmonary artery to the aorta. His pulse rate then began to rise and moist sounds appeared throughout his lungs and he began to cough up

copious, frothy sputum. Despite treatment his condition became worse and he died twenty-four hours later. In the second case an end-to-side subclavian-pulmonary anastomosis had been completed in a baby aged 2 without event, but soon after the endotracheal tube was removed, the trachea filled with frothy mucus and the baby became cyanosed. Moist sounds were heard all over the lungs and despite intensive treatment with tracheal aspiration, pressure mask breathing, venesection and administration of cardiazol and digoxin, the child died two hours later without leaving the operating room.

The remaining post-operative death followed tracheotomy for tracheal obstruction. Three children on whom we had operated for congenital heart disease required tracheotomy within ten days of operation for tracheal obstruction. The second one was almost moribund when a hasty tracheotomy was done. Six days later haemorrhage occurred into the trachea and the child was suffocated by blood. At that stage we thought that these tracheal obstructions might have been due to some trauma by the endotracheal balloon tube. When the third case occurred a bronchoscope was passed and what appeared to be a diphtheritic membrane was seen immediately below the cords. A smear was not made but we found that there was a diphtheria carrier in the ward and it is assumed that all these patients had suffered from a mild tracheal diphtheria.

#### CARDIAC FAILURE.

Seven patients on whom anastomosis has been done have had attacks of cardiac failure since operation. Three of them have died.

A.P., aged 7, had a pulmonary-aortic anastomosis performed on the 10th Oct., 1947. Two months later she had an attack of influenza and this was followed by cardiac failure with a grossly enlarged liver, pleural effusion and gross cardiac enlargement. With rest in bed, digitalis and repeated aspiration of the pleural effusion she recovered and has been in excellent health for over three years. Her heart now shows only the slight enlargement expected after a systemic-pulmonary anastomosis.

J.S., aged 6 years, had a pulmonary-aortic anastomosis done on the 27th Feb., 1948. Her pulmonary arteries were minute and there was no improvement in her condition. Now, three years after operation, she is showing signs of cardiac failure.

L.F., aged 4, had a pulmonary-aortic anastomosis done on the 5th March, 1948. His condition was not improved. Towards the end of his third post-operative year, he developed pneumonia. His heart became greatly enlarged, cardiac failure rapidly ensued and he died. Autopsy showed that his anastomosis was not patent.

W.D., aged 10, had a pulmonary-aortic anastomosis done on the 16th July, 1948. Her condition appeared to be satisfactory for nearly two years and then she was admitted to hospital in cardiac failure with a very large heart and died. At autopsy the anastomosis was completely satisfactory and not larger than an average subclavian artery.

L.M., aged 5, had a pulmonary-subclavian end-to-side anastomosis done on the 16th Dec., 1948. For two years he was capable of almost normal activity and then showed signs of cardiac failure. Although his symptoms have responded to treatment, his heart is grossly enlarged and his outlook is not good.

L.C., aged 2, was a desperately sick child, deeply cyanosed, dyspnoic, and under-nourished. A subclavian-pulmonary end-to-side anastomosis was done on the 28th Dec., 1948. Improvement was dramatic and she was taken to Newcastle in good condition on the 18th Jan., 1949. In February, 1949, she had pneumonia and her condition remained very unsatisfactory until in September, 1949, she was seen by Dr. Kempson Maddox in serious heart failure. On digitalis, rest and a salt-free diet she improved rapidly and has been in splendid health since November, 1949.

B.O.B., aged 5, had a pulmonary-aortic anastomosis done on the 27th May, 1949. He returned to an orphanage in South Australia in excellent condition. Four months after operation he was admitted to hospital with cardiac failure and he died. Autopsy showed a satisfactory anastomosis and an enlarged heart.

So far we have seen failure develop in 5 out of 33 patients who had pulmonary-aortic

anastomoses and in 2 out of 52 who had subclavian-pulmonary anastomoses. This does not necessarily show that failure is more likely after the pulmonary-aortic operation because our earliest patients nearly all had pulmonary-aortic anastomoses and irreversible cardiac failure has appeared in them after a period longer than that yet survived by most of those who have had subclavian-pulmonary anastomoses. In one patient the pulmonary arteries were so tiny that any anastomosis seemed foredoomed to failure and in another who died the aortic-pulmonary anastomosis was not patent at autopsy. Failure in these 2 cases has occurred in the normal course of the disease and not as a result of any burden added to the heart by the anastomosis.

A slight, non-progressive enlargement of the heart may be expected in nearly all patients, but in our experience the degree of enlargement has borne no relation to the type of anastomosis done.

#### LATE DEATHS.

Four patients have died since discharge from hospital. Three of these deaths have been discussed under heart failure. The fourth patient came from the New Hebrides and although we have heard that she is dead we have not yet obtained any details.

#### CONDITION OF SURVIVORS.

Table 8 shows the present state of the survivors.

TABLE 8.

Time Since Opn.	Very Good	Improved	Not Improved	Died Since Opn.	Not Traced	Too Recent
Over 3 yrs	4	1	1	1	—	—
2-3 years	26	4	1	2	2	—
1-2 years	15	3	2	—	3	—
Less than 1 year	5	—	—	1	—	4
	50	8	4	4	5	4

Because our patients are widely scattered throughout Australia, it has been impossible to make a completely scientific review of all of them.

Of the 84 patients who had anastomoses performed, 9 died as a result of the operation. Four have been too recently done to justify assessment, and 5 have not been seen or traced recently. Sixty-six remain for assessment and of these we have classified 50 as very good results. Eight are improved and 8, including 4 late deaths, are classed as not improved.

Many of the patients have been under constant observation with regular clinical, radiological and haematological assessment. The parents of the others have answered a questionnaire. Those classed as very good are capable of living a normal life in the family. They go to school or to work and play games and do all the things that normal family life entails and do not show noticeable cyanosis or clubbing of the fingers. Most of them do show some distress or slight cyanosis on extreme exertion. It must be realized that these operations do not restore the heart to normal but they do make it possible for many hopelessly crippled children to live a normal family life with little restriction of necessary activities.

Patients classed as improved have a lot more exercise tolerance than they had before operation but are still somewhat restricted. They may still be slightly cyanotic and clubbing has persisted in some of them. All of their parents state that the operation has been well worthwhile.

Patients classed as not improved include the 4 who have since died. Another one was suffering from the Eisenmenger complex and an anastomosis was deliberately carried out despite a pressure of 550 mm. of water in the pulmonary artery. Another child had congenital hydrocephalus and was mentally deficient and operation was done in the hope that she would be more manageable for her parents. The seventh patient in this group had very small pulmonary arteries and although a satisfactory anastomosis was completed, no improvement followed. She is now showing signs of cardiac failure. Operation in the eighth patient failed because of technical difficulties. She was an early case with a right-sided aorta. An end-to-side

anastomosis was begun under great tension and, because sutures were cutting out, the pulmonary artery was divided and the anastomosis completed. It was not very satisfactory and no improvement resulted. With more experience we would have done an end-to-end anastomosis in this case. Two years after operation she developed pulmonary tuberculosis and is at present in a sanatorium.

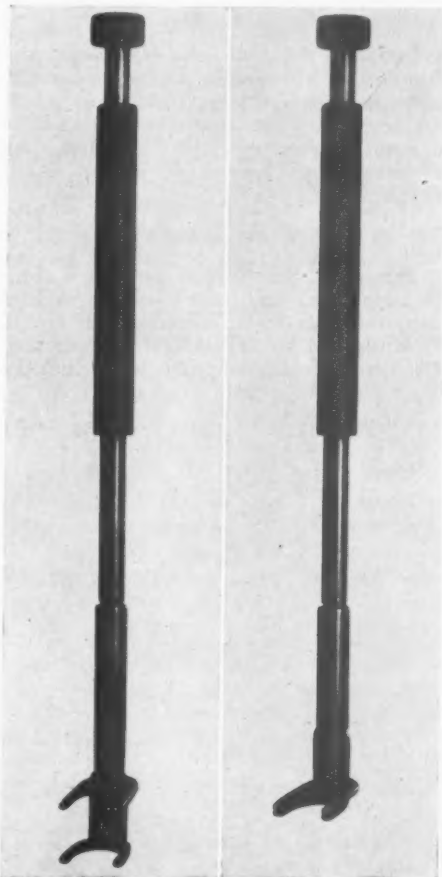


FIG. IX. Photograph of the spring-clamp used to control the pulmonary artery when performing a sub-clavian-pulmonary artery anastomosis. It is shown in the open and closed position.

#### APPENDIX.

The clamp illustrated in Fig. IX was devised to give better control of the pulmonary artery in performing a Blalock's

operation. It is easily applied and easily removed and its use enables an assistant to approximate the pulmonary artery and the cut end of the subclavian artery in all cases, so that if an anastomosis is possible it can be made without tension. We use a modified Blalock's pulmonary artery clamp to control the subclavian artery and tighten the screw just sufficiently to occlude the vessel. It has never damaged the vessel wall.

#### SUMMARY.

Two hundred and three operations performed on patients suffering from congenital abnormalities of the heart and great vessels are reviewed. The conclusions reached as a result of these operations are given. An assessment has been made of the results obtained.

#### ACKNOWLEDGEMENTS.

Success in this field of surgery is made possible by efficient team work and we have been fortunate in the association of Dr. R. H. Orton and Dr. H. B. Kay with our unit. Dr. Orton has administered the anaesthetics

to most of these patients and to his skill we attribute an almost complete absence of cardiac disturbances during operation. Dr. Orton made the first pulmonary artery clamp, and several other instruments we have used, in his own workshop, and his technical skill and physiological knowledge played an important part in this work.

Since 1948, Dr. Kay has been responsible for the diagnosis and selection of patients for operation and in association with the radiological department and the clinical research unit has developed the techniques of cardiac catheterization and angio-cardiography.

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## SUBTOTAL PAROTIDECTOMY FOR MIXED SALIVARY TUMOUR.\*

By HOWARD H. EDDEY.

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ANY operation designed to remove a mixed salivary tumour of the parotid salivary gland must satisfy two criteria; (I) removal of the tumour must be complete, and (II) the facial nerve must not be damaged.

Subtotal parotidectomy fulfils the first criterion in that an adequate amount of normal tissue surrounding the tumour, which is almost always situated in the superficial lobe of the gland, is removed. The second criterion can be satisfied if the operator possesses an accurate knowledge of the surgical anatomy of the facial nerve in relation to the parotid gland and if a technique is followed which ensures early and constant visualization of the nerve and its branches.

The object of this paper is to describe a method of subtotal parotidectomy which will, with ease, avoid damage to the facial nerve.

### ANATOMY.

Grégoire (1912) was the first anatomist to describe a superficial and a deep lobe of the parotid gland. He stated that the isthmus joining the two lobes was situated above the facial nerve and its temporo-facial and cervico-facial divisions.

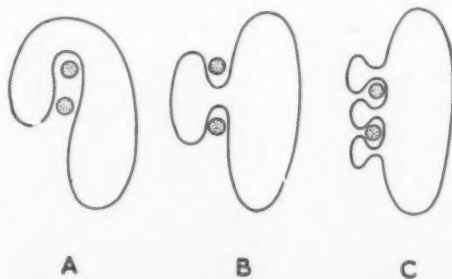


FIG. 1. The relation of the main divisions of the facial nerve to the lobes of the parotid gland according to (A) Grégoire, (1912), (B) McWhorter, (1917), and (C) McKenzie, (1948).

McWhorter (1917) also described two lobes of the parotid gland but in his opinion the isthmus joining the superficial and deep lobes lay between the main divisions of the nerve. This work was confirmed by McCormack, Cauldwell and Anson in 1945. These workers dissected 100 parotid glands and besides describing an isthmus lying between the two primary divisions of the nerve, they found that there was a great variation in the method of branching, varying in complexity, and they pointed out that anastomosis between the branches from the temporo-facial and those from the cervico-facial division was common.

Hurford (1946) dissected 11 normal adult parotid glands. In 10 of the 11 specimens the gland was bilobed. In 7 of these bilobed glands there was a well-defined isthmus and in 3 it was ill-defined. Of the 10 bilobed glands, in 4, the branches of the nerve remained strictly in the plane between the lobes; in 5, one or more branches pierced the deep lobe and in one, some of the branches traversed part of the superficial lobe.

McKenzie (1948), following the dissection of five adult parotid glands, stated that there were several isthmuses connecting the superficial and deep lobes. The branches of the nerve passed between these isthmuses so that the superficial and deep lobes of the gland may be joined through any gap in the plexus of the facial nerve. (Fig. 1.)

The preceding descriptions of the anatomy of the facial nerve in relation to the parotid salivary gland followed dissections in the cadaver. However, as a result of the performance of subtotal parotidectomy for mixed salivary tumour by the method to be described, further details of the surgical anatomy become evident.

\*Read at the Annual General Meeting, Sydney, June, 1951.

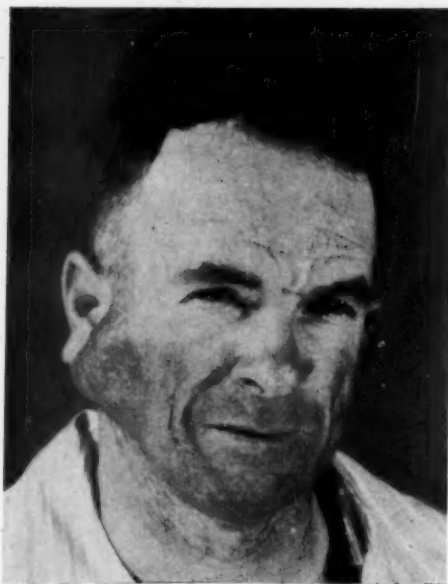


FIG. II. Photograph of a patient (case 7 of series) showing the tumour—anterior view.



FIG. III. Photograph of the same patient as in Fig. II showing the tumour, postero-lateral view.

The facial nerve enters the parotid gland on the same level as the anterior aspect of the cartilage of the external auditory meatus and just below its junction with the bony meatus. To reach the point of entry into the gland the nerve passes forwards, outwards and slightly downwards from the stylomastoid foramen, lying superficial to the styloid process. The distance between the stylomastoid foramen and the point of entry into the gland is variable but is usually about 1 cm., and before entering the gland, the nerve gives off its posterior auricular branch and the nerve to the stylohyoid and digastric (posterior belly) muscles. At its point of entry the nerve is completely surrounded by the parotid gland and gland tissue continues to surround the nerve until it divides into temporo-facial and cervico-facial divisions, about  $\frac{1}{3}$  cm. from the point of entry into the gland. To expose the point of division, gland tissue must be divided above, lateral to and below the main trunk of the nerve.

As the divisions of the nerve separate, an isthmus, which connects the superficial and deep portions of the gland, is seen between the divisions and this isthmus lies

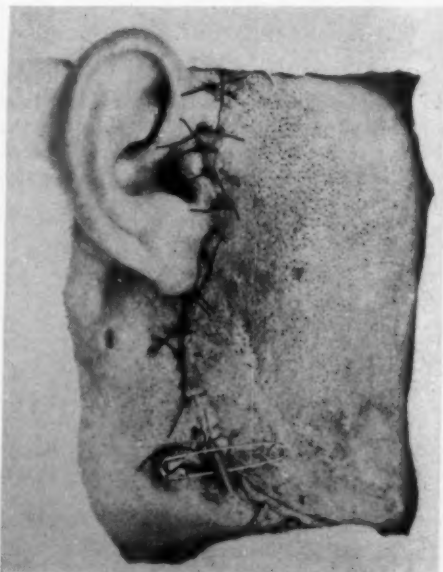


FIG. IV. Photograph showing the incision, after suture.

directly behind the ramus of the mandible. However, to bring this isthmus into view, gland tissue, which connects the superficial and deep lobes, must be divided above and below the main divisions of the nerve. Therefore to expose the nerve between its point of entry into the gland and where the temporo-facial and cervico-facial divisions pass over the ramus of the mandible, three isthmuses must be divided; one above, one between and one below the main divisions of the nerve. Divisions of the upper and lower isthmuses must commence as soon as the nerve enters the gland; the central isthmus is divided after the two main divisions of the nerve are exposed.

The branches of the two primary divisions as they pass over the masseter muscle lie in a fibrous tissue layer on the posterior aspect of the superficial lobe and this layer must be divided along the course of the branches to allow them to fall back on to the masseter muscle. In addition several small twigs to the gland itself must be divided to free the branches completely.

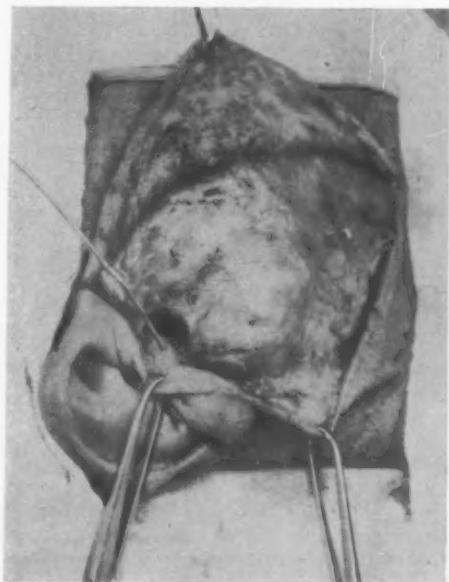


FIG. V. Photograph of the wound at operation:  
1. the skin flaps have been mobilized,  
2. a metal clip has been placed over the angle of the mandible, and  
3. a hole has been made anterior to the cartilage of the external auditory meatus, the cartilage being indicated by a probe.  
In Figs. V-XI the patient is lying on his back.

### THE OPERATION.

With the patient under endotracheal anaesthesia, 30 cc. of 1 in 60,000 adrenalin solution is injected along the line of the incision and under the skin covering the gland. This allows mobilization of the skin flaps with little bleeding and the further use of this solution in the deeper layer of the dissection obviates the necessity of ligating the external carotid artery.

The incision begins over the posterior root of the zygoma, descends in front of the external auditory meatus, extends behind the angle of the mandible and then passes forwards  $\frac{1}{2}$  inch below the mandible for a distance of 3 to 4 cm. (Fig. IV). The anterior skin flap is mobilized to expose the whole of the superficial surface of the gland. The posterior skin flap is mobilized to outline the posterior limit of the gland with its contained tumour and the anterior border of the sternomastoid muscle (Fig. IV).

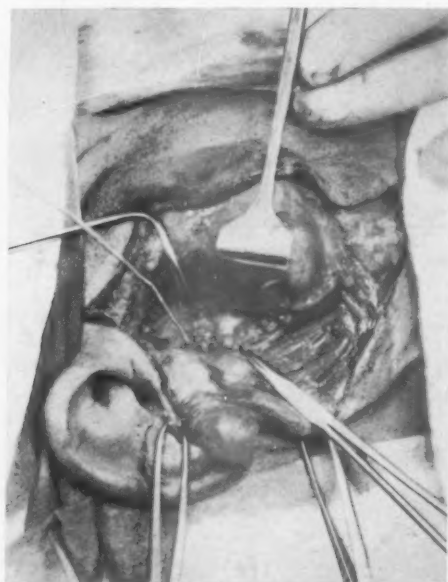


FIG. VI. Photograph of the operation showing:  
1. the facial nerve exposed 5 mm. below the spike on the inner end of the cartilage of the external auditory meatus, the spike being indicated by a probe, and  
2. parotid gland tissue lying above, lateral to and below the main trunk of the nerve.

The handle of a scalpel is then pushed medially in front of the cartilage of the external auditory meatus and a gap is opened up between the cartilage and the gland (Fig. V). On the inner end of the cartilage is a small spike which is directed slightly forwards and downwards and the facial nerve lies five to six mm. directly below this spike and slightly deeper than it. The nerve in this position is completely surrounded by the parotid gland but little gland tissue needs to be divided to expose the nerve (Fig. VI). Because of the traction on the gland necessary to expose the main trunk of the nerve at this site it must be remembered that the nerve becomes directed outwards rather acutely at this stage of the dissection.

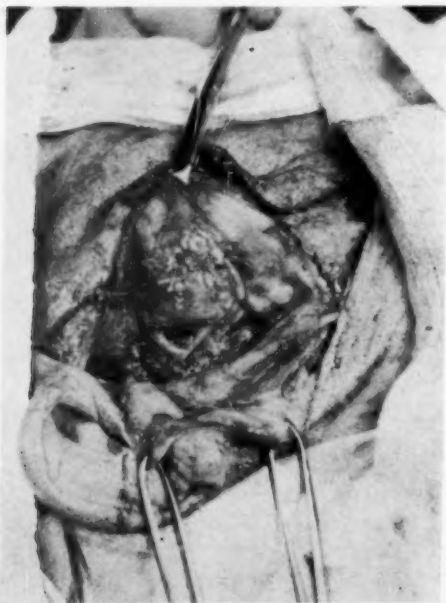


FIG. VII. Photograph of a stage of the operation:

1. the temporo-facial and cervico-facial divisions of the nerve are identified,
2. a small branch from the temporo-facial division passes directly to the gland, and
3. parotid gland tissue lies above the nerve and its temporo-facial division, between the two divisions and below the nerve and its cervico-facial division; gland tissue has been divided at these levels.

To expose the temporo-facial and cervico-facial divisions of the nerve parotid gland tissue has to be divided above, lateral to and

below the main trunk of the nerve. As the main divisions of the nerve are traced forward, further gland tissue is divided above and below the divisions, and in addition between the divisions (Fig. VII). When the dissection reaches the posterior border of the ramus of the mandible, the superficial lobe has been completely detached from the deep lobe by the division of three isthmuses, one above the main trunk of the nerve and its temporo-facial division, one between the temporo-facial and cervico-facial divisions and one below the main trunk and its cervico-facial division (Fig. VIII). The deep lobe then lies beneath the main trunk and its primary divisions and in this portion of the gland the posterior facial vein lies (Fig. XI).

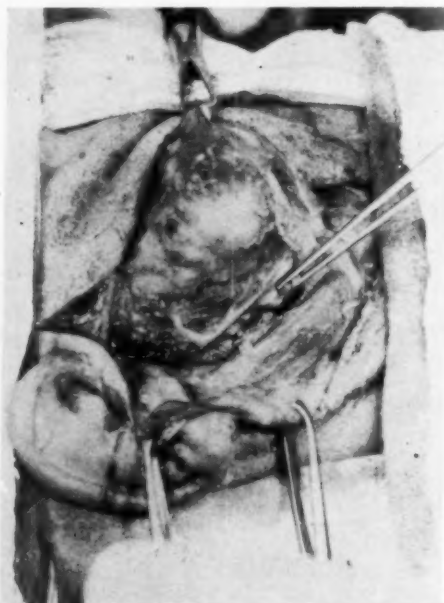


FIG. VIII. Photograph of a further stage of the operation showing:

1. further mobilization of the superficial lobe,
2. the buccal branch arising from the cervico-facial division of the nerve, and
3. dissecting forceps identifying gland tissue which has been divided below the cervico-facial division of the nerve.

The main divisions of the nerve and the branches of these divisions are detached from the superficial lobe by dividing a

firm fibrous tissue layer covering the posterior aspect of the superficial lobe along the course of these branches. In addition several small twigs to the gland itself must be divided (Fig. IX). The operation is completed by dividing the parotid duct at the anterior border of the masseter muscle (Figs. X and XI).



FIG. IX. Photograph of a further stage of the operation showing:

1. the superficial lobe partly dissected off the masseter muscle,
2. the branches of the nerve lying on the masseter muscle after their mobilization from the fascia on the posterior surface of the superficial lobe, and
3. a probe identifying a long twig to the gland from the zygomatic branch of the temporo-facial division.

### RESULTS.

Subtotal parotidectomy for mixed salivary tumour, following the technique described, has been performed on nine occasions without any resultant permanent facial paralysis (Figs. II, III, XIII and XIV).

In one patient who had a recurrence of a mixed salivary tumour, one branch of the cervico-facial division of the nerve had to be divided as it passed through scar tissue from the previous operation. This resulted

in a temporary paralysis of the depressor anguli oris muscle but, because of the anastomosis between branches of the temporo-facial and cervico-facial divisions of the nerve, this paralysis disappeared. In one case occurring early in the series, a complete facial nerve paralysis resulted; this was regarded as due to excessive traction on

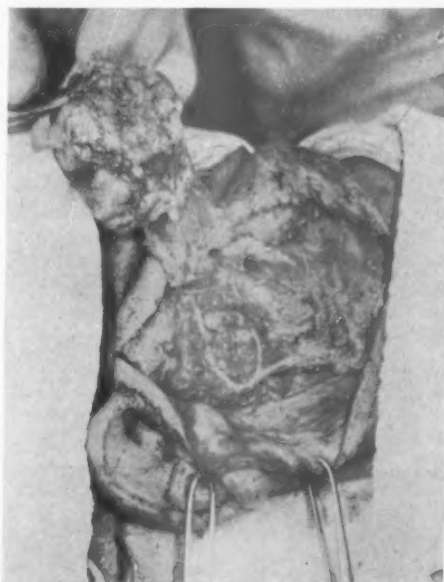


FIG. X. Photograph of a further stage of the operation showing:

1. the superficial lobe completely mobilized with the parotid duct and its surrounding fascia alone remaining to be divided, and
2. the branches of the nerve lying on the masseter muscle.

the nerve in the early stages of the dissection. With appropriate splinting of the facial muscles, this patient recovered completely from this paralysis in three months.

No patient developed a salivary fistula and no case of Frey's syndrome has been observed.

The operation described can be extended to remove portion or all of the deep lobe, if this lobe be involved by tumour growth. This has been done on two occasions, once

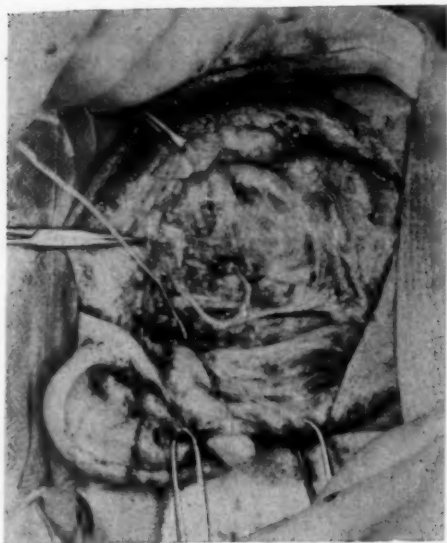


FIG. XI. Photograph showing the operation completed:  
1. the spike on the cartilage of the external auditory meatus is identified by a probe, and  
2. the deep lobe of the parotid gland lies above the nerve and its temporo-facial division, between the divisions and below the nerve and its cervico-facial divisions.



FIG. XII. Photograph of the specimen after removal. It shows the tumour surrounded by normal glandular tissue.

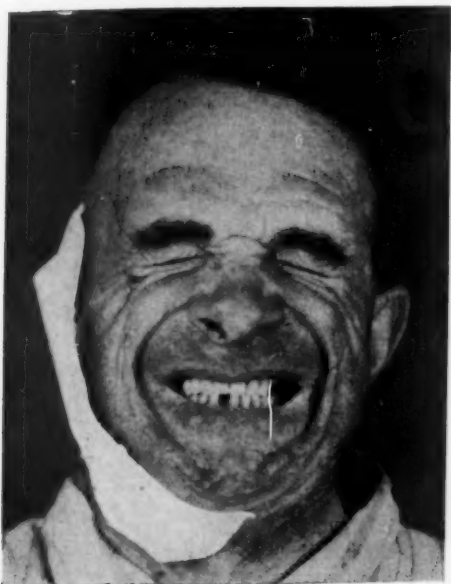


FIG. XIII.  
Photograph of the patient three days after operation, showing good contraction of the facial muscles and hence integrity of the nerve.

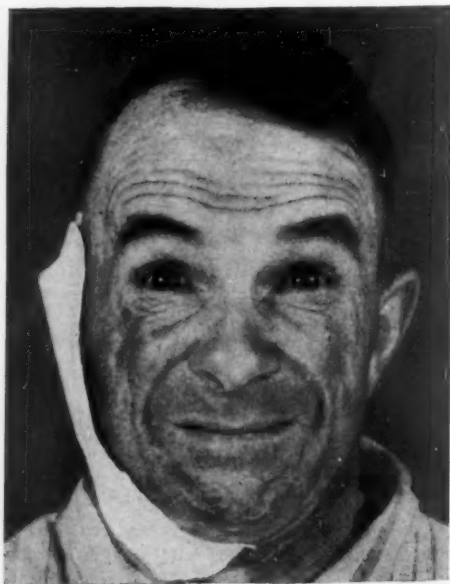


FIG. XIV.

for a carcinoma of the gland and once for a recurrent mixed salivary tumour. With early and constant visualization of the facial nerve and its divisions and branches such an operation may in some cases avoid any damage to the facial nerve. However, more often than not, it will be found necessary to divide one or two branches of the cervico-facial division of the nerve and this was done in the above two patients in order to remove the tumour.

#### SUMMARY.

A technique for removal of the superficial lobe of the parotid salivary gland, with early and constant visualization of the facial nerve, is described. Some points in the anatomy of the facial nerve in relation to the parotid gland are discussed.

#### ACKNOWLEDGEMENT.

The photographs of the operation are the work of Mr. R. Inglis, Clinical Photographer at the Royal Melbourne Hospital and to him I am greatly indebted.

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## ABDOMINAL EXPOSURE.\*

By LEO DOYLE.

Melbourne.

IN recent years the subject of abdominal exposure has come to mean more than the possible length of a vertical incision. The design of a proper exposure requires a contemplation of the anatomical parts involved in the proposed operation and a consideration of the various factors required to display them adequately whilst doing the least amount of harm. These factors are: The anaesthesia, the position of the patient on the operating table, the lighting of the operation area, the incision used (probably the most important single factor) and the amount and type of retraction employed.

Of recent years developments in anaesthesia have resulted in the achievement of a degree of muscular relaxation that was known formerly only to those of us who were accustomed to use spinal anaesthesia. The use of antibiotics has relieved us almost completely from the fear of sepsis, thus freeing a large wound from the dangers of infection spreading through extensively opened tissue planes. Knowledge of the factors concerned in wound healing enables us to remedy the deficiencies responsible for its occasional failure, and so removes another factor that might make us diminish the length of the incision. Also the use of non-absorbable suture material will keep a large wound together until it has healed.

The knowledge that a cut muscle will heal frees us from the obligation of trying to make as much as possible of our incision by simply splitting muscle fibre; we know that a cut nerve is much more important than a cut muscle. I have been leading up to a statement that incisions should be liberal but not necessarily large. They must be large enough, however, so that the surgeon, using the equipment he is accustomed to, will have adequate room to deal with the disease found, and they should be made with as little nerve damage as possible.

Lighting is a most important part of the exposure, since a viscus may be just as invisible from lack of light as from a too

small incision. Daylight is no longer considered adequate; some form of focussed lighting is a necessary adjunct, so that that which has been uncovered may be fully illuminated. Recently, so I have been informed, the customary method, in which there is one central focussing light which may or may not be moveable, has been superseded by the use of several such lights which are attached to moveable pedestals running on the floor into any convenient and handy position. I should imagine that this would have advantages over our present system.

Position of the patient on the table is most important and, of course, varies with the different organs to be attacked. The usual supine position serves for many cases, and I do not have to emphasize the advantages of the head down position in a pelvic operation; but what some may not always remember are the advantages of the reverse Trendelenberg position in operations on the upper abdomen or the advantages to be gained when attacking the ascending or descending colon by so rolling the table that the area operated on is uppermost. With an adequate incision and efficient retraction the use of a rest which forces the spine into a position of hyper-extension becomes unnecessary; so, some post-operative pain is avoided.

Of recent years the ease and safety with which the formerly sacrosanct pleural space may be invaded has simplified the approach to the organs placed immediately under the costal cage. This has made the cardiac end of the stomach almost as easy of approach as the pyloric end and has made Lahey's (1950) plea for a total gastrectomy in all cases of carcinoma of the stomach feasible and reasonable. The difficult, or even otherwise, impossible, nephrectomy becomes almost easy when the peri-renal space is approached by removing the 10th or 11th rib and then carrying the incision across the rectus abdominis muscle.

\*Read at the Annual General Meeting, Sydney, June, 1951.

A further advance, the result of a realization that a cut muscle heals and a cut nerve does not, is the tendency to make incisions between nerves and across muscle; the resultant has been usually a transverse incision. There is no doubt that a vertical incision in the hands of an expert is capable of giving a wide access but there is no doubt

that as one goes on making transverse incisions one finds gradually that the occasions, in which its use is considered helpful, become more and more frequent.

Before going on to consider the various methods of exposing different abdominal organs, attention must be called to the fact that exposure is but a part of a surgical procedure and that an operation associated with an adequate exposure will be a failure unless all other details of the operation are sufficiently cared for.

In discussing the various incisions I do not propose to mention the paramedian incisions, except to say that on some occasions a better incision is not available; which does not mean that I think they should be used as a routine unless a different incision is necessary. The paramedian incision should be used on its merits.

I have on several occasions credited to Rutherford Morison the aphorism "There are only two incisions in the abdomen — straight up and down in the midline, or straight across anywhere else"; and this more or less agrees with my own views. Here I might say that the long thin belly is probably better approached through a vertical incision and the broad squat one by way of a transverse one.

In designing an incision, the layout of the abdominal intercostal nerves (Fig. I) must

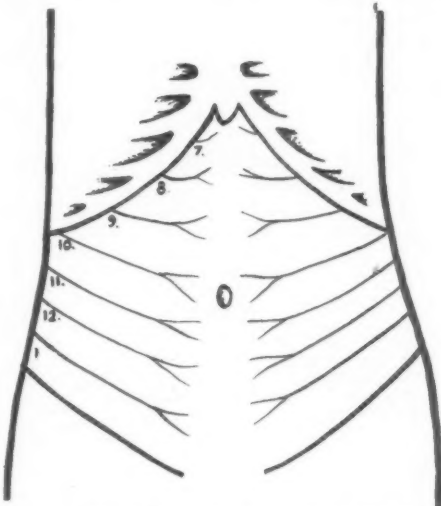


FIG. I. Drawing to show the general distribution of the nerves in relation to the anterior abdominal wall.

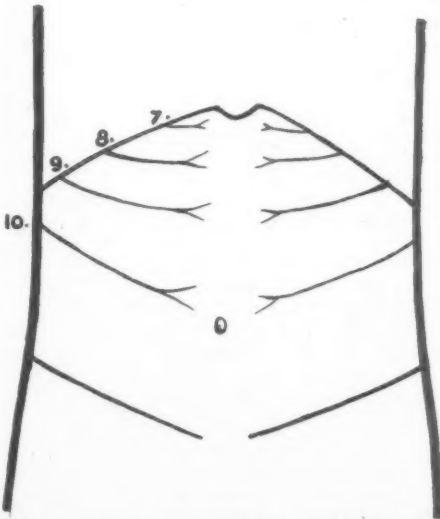


FIG. II. Diagram to show the broad abdomen with wide costal margin; this invites a transverse incision.

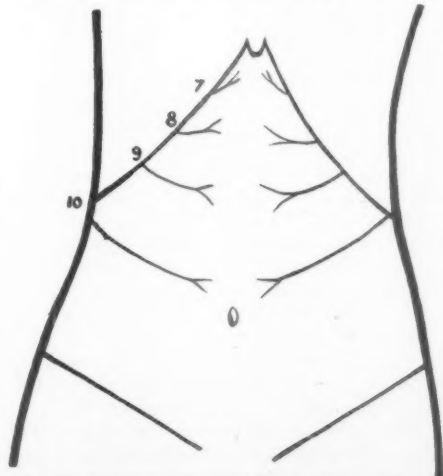


FIG. III. Diagram of anterior abdominal wall with narrow costal margin; this requires a vertical incision.

be studied, and an attempt made to design a suitable incision which will damage none; however, one abdominal nerve can be cut across without doing any great harm.

A diagram of the intercostal nerves will show how a transverse incision (Fig. II) is better in a broad individual, and a longitudinal one (Fig. III) better in the long thin type. This statement is more accurate of upper abdominal incisions; it is rare for the pelvis to be narrow.

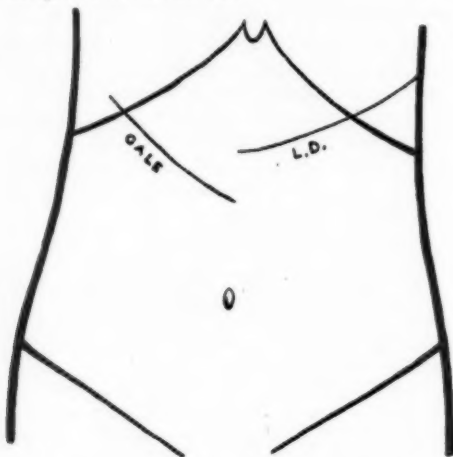


FIG. IV. Diagram of anterior abdominal wall showing the Gale and Doyle incisions. Note that the Doyle incision goes further on to the chest wall.

In attacking the upper abdomen it is at times profitable to extend the incision into the chest by going across the costal margin. Many years ago in conjunction with Mr. A. Rowlands, who was Acting-Professor of Anatomy in Melbourne at the time, I designed an incision (Fig. IV) which would enable the costal margin to be cut without encroaching on the pleural cavity. This incision depended on the facts that the diaphragm and transversus abdominal muscles are closely intermingled under the costal margin (Wood Jones says they are only one muscle) and that the pleura can be stripped off the diaphragm in much the same way as a hernial sac is stripped out. Mr. Charles Gale of Geelong (1949) has recently published an incision which is much the same but he does not cut the costal margin. Incidentally I think it might be said that, if there are no disqualifying factors such as poor vital capacity or other conditions which

might render the oxygenation of the blood suspect, it is safe to open one pleural cavity without tracheal intubation. However, we need not worry about this as with correct anaesthetic methods we can now open the chest freely. This can be done by cutting across the costal arch, and then either by going between two ribs or better still by excising one. It can also be done according to Wangenstein (Fig. V) — who was commenting on a paper by Lahey (1950) on the subject of total gastrectomy—by splitting the sternum and cutting outwards extra-pleurally on to the 4th left interspace.

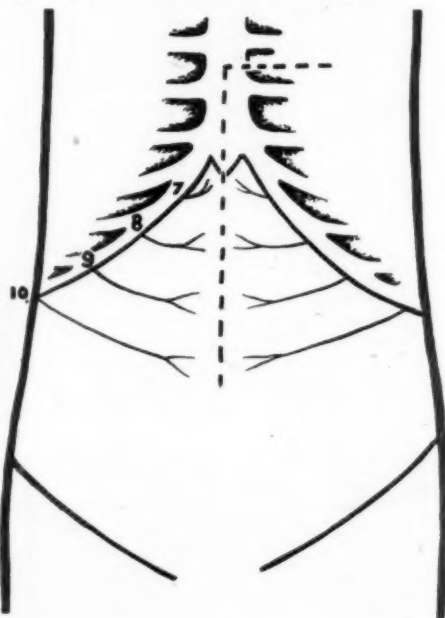


FIG. V. Diagram of the anterior chest and abdominal walls showing the incision advocated by Wangenstein.

In opening the abdomen for a high carcinoma a subcostal incision running out to the 8th or 9th rib gives an exposure which enables one to determine the operability of the tumour. If this incision is then continued up in the rib bed after excision we obtain a wider exposure, and need cut but one nerve. To remove a stomach for duodenal ulcer, as the associated scarring will frequently pull the duodenum well down towards the paracolic gutter, the incision should be over the duodenum, well on the right side (Fig. VI) and sufficiently far over to the left side

to enable the desired amount of stomach to be removed easily. There is seldom any necessity to open the chest in a gastrectomy which is being done for peptic ulcer.

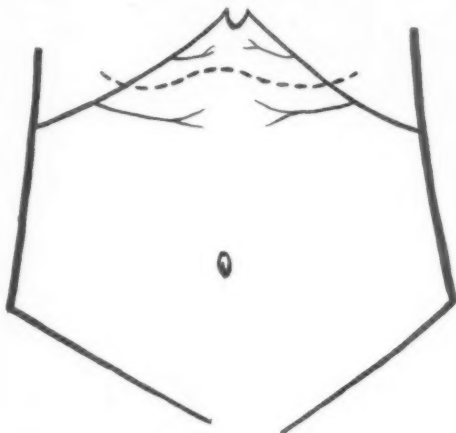


FIG. VI. Diagram of the anterior abdominal wall showing the incision in the upper part of the abdomen used for subtotal or 70 per cent. gastrectomy.

It is possible and useful on occasion to extend a vertical incision by cutting out along an intercostal nerve from the lower end of incision (Fig. VII).

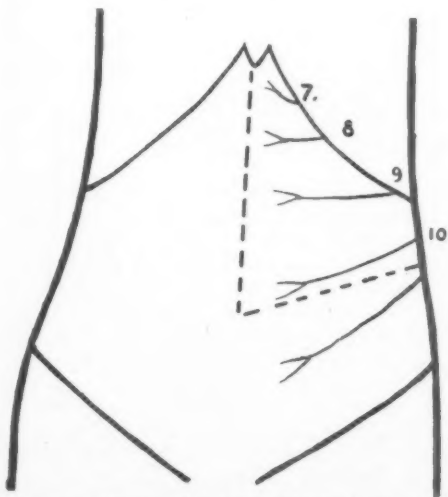


FIG. VII. Diagram of anterior abdominal wall showing a midline vertical incision prolonged by a transverse incision.

For the operation of cholecystectomy a subcostal incision gives adequate exposure. The incision runs under the costal margin, starting against the costal margin of the left side and passing outwards to the outer border of the rectus abdominis, and then turning into the costal margin (Fig. VIII). It runs practically parallel to the 8th intercostal nerve; perhaps between it and the 7th. If it becomes necessary to go further laterally the 8th nerve can be cut and the incision prolonged in that direction. By the correct use of a self-retaining retractor this incision will give good access to the gall bladder without the use of any gall bladder rest. A good trick here is to put in a Devine frame to open the incision and then substitute mechanical hands for three of the blades, using the Fritsch blade on the costal margin and three mechanical hands to hold the packed off intestines out of the field. This trick can be used for most incisions, the mechanical hand substituting for the assistant's hand. The mechanical hand has the advantage that it is smaller and does not get tired.

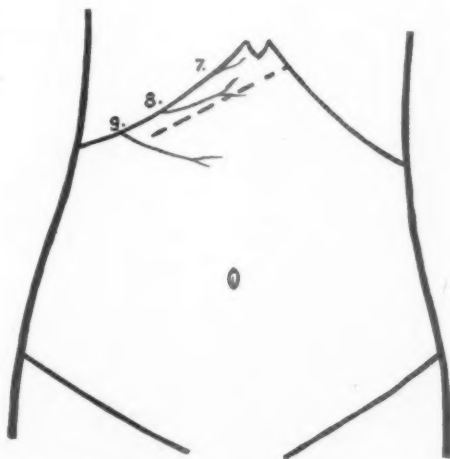


FIG. VIII. Diagram showing incision for cholecystectomy.

Treatment of post-operative stenosis of the common bile duct provides a most strenuous surgical exercise, generally because the condition itself is difficult to treat but also because usually it has to be approached through a welter of adhesions that bleed

freely. I have a feeling, in secondary operations, that it is a good idea to try and break in where there are no adhesions and then advance into the affected area. With this in mind, when a Kocher or subcostal incision had been made, I would approach the lesion either by a long right rectus incision that would take me into a clean para-colic gutter, or, if the patient were broad-bellied, I would make a thoraco-abdominal incision and in that way get into a clean field.

A transverse incision from near the anterior superior iliac spine to the umbilicus (Fig. IX) is very good for exposing a carcinoma of the caecum or for dealing with any disease presenting difficult technical problems in this region. The incision can be prolonged by adding a vertical midline prolongation upwards or downwards.

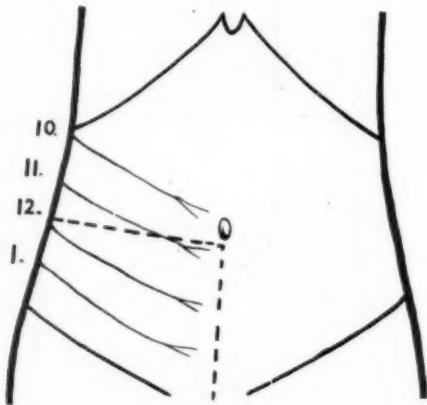


FIG. IX. Diagram showing an incision for lesions in the right iliac fossa.

Why surgeons, who propose to make a McBurney's muscle splitting incision (Fig. X), make their skin incision parallel to the fibres of the external oblique muscle is incomprehensible to me. A transverse incision, or rather one that fits in with the skin lines, gives equally good access and leaves an excellent scar, whereas the scar of the other incision is always ugly.

Access to an acutely inflamed appendix should be so liberal that there will be no danger of its being ruptured by force during delivery. Additional access may be obtained by cutting the transverse and internal oblique muscles parallel to the split in the external

oblique tendon and carrying this outwards as far as necessary. A transverse incision can be extended across the rectus abdominis to the midline or further.

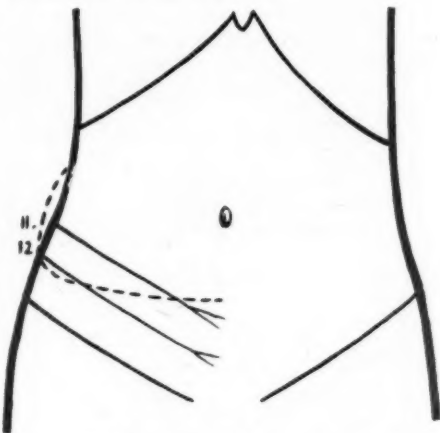


FIG. X. Diagram showing the incision usually employed for appendicectomy with a medial and vertical prolongation.

Sometimes, when the tip of a latero-caecal appendix is too high for comfortable removal or when one has encountered an undescended appendix, satisfactory access can be gained by prolonging the split in the external oblique muscle to a higher level and then making a second split in the internal oblique and the transverse muscles.

The common lesion of the other side of the abdomen (the left colic carcinoma) is dealt with either by a left transverse incision similar to that just described (Fig. XI), or by a vertical midline incision.

This vertical incision can be extended if necessary outwards towards the tip of the 10th costal cartilage. This is a very useful manoeuvre if it should become necessary to free the descending colon, and possibly the splenic flexure, in order to get enough colon to perform an anastomosis. This incision is also very useful in carrying out a total colectomy, as the difficult area in this operation is the splenic flexure. Usually an incision that gives access to the distal part of the pelvic colon will not allow it to this structure. This incision, though wholly on the left side, allows quite a good access to the hepatic flexure. Its transverse limb can, of course, be prolonged across the other rectus muscle.

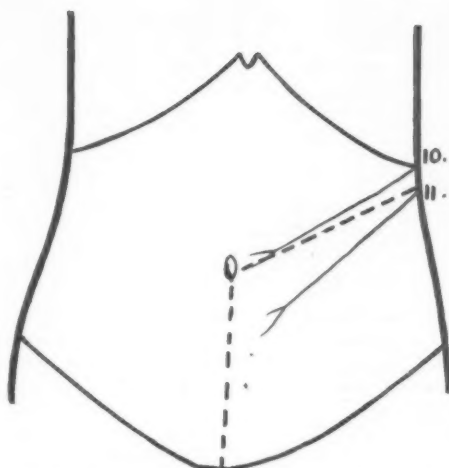


FIG. XI. Diagram showing incision for lesions of the descending and sigmoid colons.

For the operation of total cystectomy with simultaneous transplantation of the ureters a very good exposure can be obtained by doing just this, and combining these two incisions (Fig. XII), that is, making a transverse incision, from just above and medial to the anterior superior iliac spine on the right side to a similar position on the left side, passing just below the umbilicus and then making a vertical limb in the middle line down to the symphysis pubis. This wound is extensive but heals well, and the patient is remarkably free from post-operative pain.

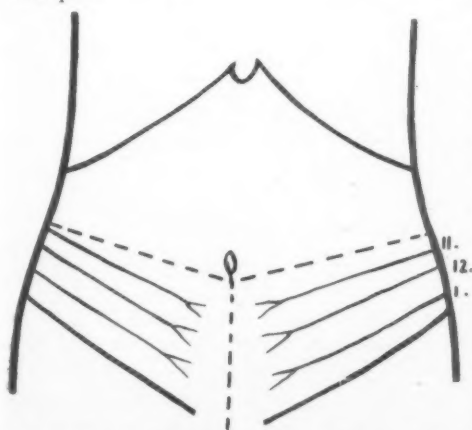


FIG. XII. Diagram showing incision for total cystectomy with bilateral ureteral transplantation.

### Hernia.

I can never understand why, when operating for this condition, surgeons will make an incision parallel to the fibres of the external oblique tendon. A transverse incision or one in the folds of the skin (Fig. XIII) will give equally good exposure and heals without the ugly scar that is such a common feature of this operation.

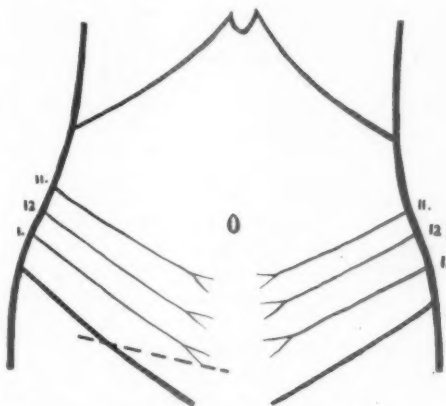


FIG. XIII. Diagram showing the skin incision of operation for inguinal hernia.

### Renal Exposure.

The kidney, even when normal in size and position, is to some extent under the costal cage and, when it is enlarged, can be much more so. One of the usual kidney incisions will give reasonable access to a kidney that is of normal size and not adherent to adjacent structures. If it is desired to ligate the pedicle of a tumour-containing kidney or if it is thought that, owing to adhesions, the pedicle will be difficult of access, the approach should be across the diaphragm. In operating for cortical tumour of the adrenal this approach is even still more necessary as it is important that such a tumour be removed without being squeezed or pressed; such a removal attempted from below the ribs is impossible.

I have spoken against a gall-bladder rest but I believe that a kidney rest is a good instrument. However, it should be of the type whose height is varied by a handle so that the most suitable degree of elevation can be maintained. Using one of these with a Devine retractor I have found on occasion

that the access to and mobilization of the kidney was improved by placing the frame in position whilst the rest was elevated and then lowering the rest almost to table level for the completion of the operation.

It is desirable to say something about retraction and retractors. Anyone who has used local anaesthesia to any extent is aware of how controlled and gentle retraction must be if harmful stimuli are to be avoided and that every pull hurts. Even with the very best assistants manual retraction must be a series of pulls, each of which hurts, rather than a steady continuous one which soon ceases to hurt. A self retaining retractor (I prefer the Devine—I suppose because I am used to it) gives a steady traction which does not cause a series of harmful stimuli to be initiated in the wound. A very distinguished English surgeon who once paid me the compliment of watching me operate said as I put the frame of the retractor into position, "I call on heaven to witness that old as I am I have

never yet put a self-retaining retractor into any patient's wound." Now I think that remark was silly, just as any statement of "always doing this" or "never doing that" is bound to be silly. Don't, however, imagine you can go to an instrument maker, buy such a retractor, and that all your troubles will be over. You have to learn to use the instrument and this process will take quite a while and until you have an extensive familiarity with it you are not entitled to say it is good or bad.

In conclusion, good abdominal exposure is the product of many factors, and implies more than just a long incision; also, as one becomes accustomed to it, a transverse incision seems to give the best access whilst doing the least harm. Lastly there cannot be a good exposure without good anaesthesia.

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# TUMOURS AND CYSTS OF THE MEDIASTINUM.\*

## PART ONE.

By KONRAD HIRSCHFELD.

*Brisbane.*

THE traditional picture of a mediastinal tumour—a patient propped up in bed, with a dusky swollen and congested face, an anxious expression, and a brassy productive cough as distressing to observers as to himself—is, fortunately, now becoming rare. For, as a result of the numerous mass radiological surveys of large sections of the population, these mediastinal tumours are diagnosed early at a stage when they are symptomless. These surveys, originally undertaken with a view to the discovery of subclinical tuberculosis, are yielding as a by-product numerous subclinical cases of other thoracic and cardiac disease. Brewer and Dolley (1949) state that Prows reviewed 2,821 twenty-one millimeter chest films taken as a mass survey on routine admissions at a private hospital in Los Angeles and that there were 5 cases of probable pulmonary tuberculosis and 5 cases with probable mediastinal tumours. As these patients had already been screened to some extent before admission these figures do not represent the true incidence of these diseases, but do emphasize the necessity for due attention to the mediastinum in such surveys.

Mediastinal tumours which were visible but overlooked in enlistment films have been discovered locally only in discharge films of some service personnel, which emphasizes the point made by Prows.

Since the best organized and largest of the mass surveys so far are those conducted by the services whose material is mostly young adult males, the age and sex incidence of the patients with these symptomless mediastinal tumours is perhaps biased. No doubt as the mass survey method is applied to the general population the true incidence of the tumours will emerge.

As with early pulmonary tuberculosis, as thus diagnosed, there are many doctors who do not believe in active treatment of the

symptomless mediastinal tumour. They prefer to wait until they have authority for action from the onset of symptoms; symptoms which often appear at a stage at which the lesion is no longer amenable to easy and successful surgery. This situation recalls McBurney's remarks about the new approach to appendicitis when he stated that no longer need he visit a patient day after day awaiting the authority of established peritonitis before he dared take action (Cope, 1939).

A survey of the literature or a brief experience of mediastinal tumours must convince the doubter that the sooner an attempt is made to remove these tumours the more likely is a successful outcome for the patient.

The mediastinum is divided by the heart and great vessels into three compartments, anterior, middle and posterior, and the vascular structures are in the middle. The portion above the base of the heart is the superior and, that below, the inferior mediastinum. In the anterior mediastinum are found the thymus, fat, lymph nodes, vessels and nerves and occasionally thyroid and parathyroid glands or portions thereof. In the posterior compartment lie the trachea and oesophagus, vessels, lymph nodes, vagus and sympathetic nerves and the origins of the intercostal nerves and on occasion the thyroid or parathyroid glands may be found there also. The anterior boundary of the mediastinum is the sternum and costal cartilages and posteriorly the spine and the necks of the ribs.

In this paper, as in others, the subject of the discussion is swellings of the mediastinum, whether tumours or cysts, and such swellings are considered to lie in this category if they are extra-pleural and abut on the mediastinal shadow in an X-ray film made in the antero-posterior or postero-anterior projection. The terms of reference are thus somewhat loose.

\*A condensed version of this paper was read at the Annual General Meeting, Sydney, June, 1951.

This paper is based on a study of 26 cases seen in Brisbane and a brief survey of recent literature of the subject. This survey is by no means complete as sufficient library facilities are not available, but nevertheless data on 600 cases has been gathered.

Tumours or cysts in almost every structure in the mediastinum have been seen and so their numbers are legion. Most of them appear to be developmental in origin, and, although the majority are benign, many are malignant or become so.

Inflammatory lesions are not common but hydatid cysts, gummata and tuberculomata have been described.

The tumours and cysts may be classified according to origin:

- I. *Tumours and cysts of developmental origin.*
  - A. Teratomata which include dermoid cysts.
  - B. Bronchogenic cysts—also called aberrant lung cysts.
  - C. Pericardial coelomic cysts—also called spring water cysts.
  - D. Gastrogenic or enterogenic cysts also called reduplication cysts of the oesophagus.
  - E. Pleural exclusion cysts.
  - F. Cysts in which there is insufficient data for classification.
  - G. Cysts of accessory lung tissue.
  - H. Diaphragmatic herniae.
- II. *Tumours of the bony cage of the thorax.*
- III. *Neurogenic tumours.*
  - A. (i) Nerve sheath tumours—Schwannoma or neurolemmoma, also called neurinoma.
  - (ii) Neurofibroma.
  - B. Nerve cell tumours.
    - (i) Neuroblastoma—immature.
    - (ii) Ganglioneuroma—mature.
- IV. *Tumours of trachea or oesophagus.*

V. *Tumours or cysts of heart and great vessels.*

VI. *Tumours of thymus gland.*

VII. *Tumours of thyroid or parathyroid glands.*

VIII. *Tumours of connective tissue origin.*

- A. Lipoma.
- B. Haemangioma.
- C. Lymphangioma.
- D. Fibroma.
- E. Leiomyoma.

IX. *Malignant tumours.*

- A. Reticulosarcoma group or lymphomata.
- B. Secondary carcinoma.
- C. Superior sulcus tumours.

X. *Inflammatory tumours.*

- Tuberculomata.
- Gummata.
- Hydatid cysts.
- Coccidiosis.

XI. *Boeck's sarcoid.*

Although there are so many different tumours, the symptoms produced in the various types of tumour are similar and depend on the bulk and situation of the tumour and its rate of growth. Until the bulk is large there are few symptoms.

Respiratory symptoms include cough, dyspnoea, cyanosis, stridor and haemoptysis, and, if there is infection of collapsed lung, foul sputum, pyrexia, toxæmia and clubbed fingers.

Cough is often dry and irritable. It may be paroxysmal, and, if accompanied by air hunger, the patient feels as if he is about to suffocate. It appears to result either from pressure on the trachea or bronchi or from irritation of the vagus, or other nerves in the thorax.

Dyspnoea may arise from diminution of vital capacity by the bulk of the tumour; or from obstruction to the trachea or bronchi, with consequent collapse of lung. The dyspnoea, like the cough, may be paroxysmal and resemble asthma—a resemblance which is enhanced by the presence of a wheeze or

by stridor. Stridor, due to diminution of the lumen of the trachea, is a later symptom; it may be due to involvement of the recurrent nerve and is then accompanied by a brassy cough.

Haemoptysis occurs in 20 per cent. of mediastinal tumours and may be due to erosion of a vessel in the respiratory tract or secondary infection. As a cause of haemoptysis mediastinal tumour runs fourth to tuberculosis, bronchogenic carcinoma and bronchiectasis (Abbott, 1948).

Infection in the lung behind an obstruction, just as it is in bronchogenic carcinoma, is very often the first sign to the patient that anything is amiss.

Discomfort in the chest or epigastrium or the sensation of a lump or of fullness under the sternum is often the only symptom of these tumours.

Pain which may be severe and lancinating, or mild and merely a dull ache, is often paroxysmal, and frequently radiates. It may be of pleuritic type, aggravated by inspiration, and due to distension of the pleura. A dull ache, worse at night, is produced by pressure on bone. Lancinating pain is characteristic of pressure on nerves. It may radiate to the face, to the shoulder, down the arm, or into the epigastrium. Pain of an anginal type is not uncommon.

Nerve pressure may be such as to cause other disturbances of function—Horner's syndrome, alteration of the voice, gastrointestinal symptoms, or paralysis of the diaphragm.

Dysphagia is a frequent complaint and is usually worse with solids than fluids.

Pressure on the great veins produces the engorged dusky face and dilated swollen veins so well known in mediastinal tumours; it is a sign of bad omen. It is, in the early stages, only noticed during exertion or when the patient bends over. It is often found in the malignant tumours.

Tumours in the inferior mediastinum may produce few signs of disease other than an increase in dullness on percussion. Pressure on bronchi or trachea may produce changes

recognizable on auscultation as bronchial breathing. Disorders of cardiac action may result from displacement of the heart.

In the superior mediastinum signs are more easily recognized. The trachea may be displaced or compressed; the veins may be engorged; and signs of nerve involvement are frequently seen.

If the tumour is very large there may be enlargement of the chest over the tumour, and there may be tenderness of the chest wall.

Some of the mediastinal tumours, particularly the neurogenic variety, spread into the spinal cord and may produce cord symptoms as a result. These are the dumb-bell tumours, and, when such an extension is present, there is often erosion of the pedicles of the vertebrae with an enlargement of the intervertebral foramina.

A peculiarity of mediastinal tumour is that the symptoms may alter with an alteration in posture.

It is apparent that if so many of these tumours remain symptomless for a long time their early diagnosis depends on routine examination, particularly of a radiological nature. It should be unnecessary to stress that this radiological examination is incomplete unless it includes films taken in lateral and oblique projections as well as in the postero-anterior direction. Although juries have not yet found failure to take a lateral X-ray of the chest to be negligence, a time will surely come when they will.

The object of the radiological study is to visualize the lungs, the mediastinal structures and the bones, the ribs, vertebral bodies and intervertebral canals.

Erosion of bone is common with all types of tumours in the posterior area. The erosion may be regular with round areas of excavation of the bone. The bone may be thinned or narrowed. These are pure pressure effects and do not necessarily indicate malignancy. There may, however, be an irregular erosion of the bones with loss of bone substance and a pattern characteristic of malignant invasion. The ribs may be pushed apart and thus the intercostal spaces widened. The intervertebral foramina may be widened and

in all posterior tumours which extend to the ribs special care must be taken to render these foramina visible.

In addition to X-ray films, examination by the fluoroscopic screen is essential. Movement of the tumour when the patient swallows or when he breathes can thus be ascertained. The movements of the diaphragm and pulsation or otherwise of the tumour must be noted.

If there is pulsation a kymogram may enable a distinction to be made between expansile and transmitted pulsation. Body section radiography may yield further information.

Contrast radiography of every kind may be required to effect a diagnosis. Examination of the lungs by bronchography, of the oesophagus, and its relation to the tumour, by a barium swallow and of the vessels, by injection of an opaque medium, may supply essential information.

Bronchoscopy and oesophagoscopy both are indispensable investigations and yield valuable information both of a positive and a negative kind.

Diagnostic pneumothorax is not of much value, although, when combined with thoracoscopy, it is sometimes helpful. This step in diagnosis is now replaced by thoracotomy. Tuberculin reaction, Casoni test and Wassermann reaction as well as investigation of the sputum are all essential.

Trial X-ray therapy may be useful but should only be given for one month, as the only group of tumours which are radio-sensitive is the lymphoma group. This type of therapy should not be tried until all other investigations point to the likelihood that the tumour belongs to this group.

Aspiration biopsy is only justifiable in tumours likely to be inoperable as thoracotomy is no more fatal and much more informative. Biopsy of lymph nodes, if such be present, should be done.

The diagnosis of these tumours may be extremely difficult. That a mediastinal tumour is present is comparatively easy to determine provided radiographs are made in at least two planes; the nature of the tumour is another matter to decide. The

symptoms and signs are produced by location rather than by the nature of the tumour, but some tumours have definite characters which may help in diagnosis.

Areas of calcification may occur in the wall of a teratoma, of an aneurysm or of a thymoma; in an adenoma of the thyroid or in an osteochondroma.

Evidence of pressure on bone such as erosion of ribs or vertebrae is found in the neurogenic tumours, gastrogenic cysts, aneurysms, superior sulcus tumours and in osteochondromata.

In the anterior mediastinum are found the teratomata, tumours of the thymus and thyroid glands, pericardial coelomic cysts and aneurysms of the ascending aorta and aortic arch.

The teratomata may be very large and are often in the lower part of the anterior mediastinum. Their outline may be irregular or even angular and there may be a pedicle. They may show a fluid level. If they contain well developed bone or teeth the diagnosis is certain.

A thymoma is accompanied by myasthenia gravis in about 75 per cent. of cases. It is usually above the base of the heart.

Aneurysms of the ascending aorta are very like teratomata in the same location. The aneurysms should pulsate but often do not, while teratomata should not pulsate but often appear to do so. Kymograms may help in the differentiation of one from the other as the aneurysm should exhibit expansile pulsation. Nevertheless pulsation as a diagnostic sign in mediastinal tumours is but a snare and a delusion.

The pericardial coelomic cysts commonly occur at the pericardio-phrenic angles. They are round and may be multiple. They are often small and usually they do not cause any symptoms.

An intra-thoracic goitre moves when the patient swallows. It may have an obvious attachment to the cervical thyroid.

In the posterior mediastinum are found neurogenic tumours, gastrogenic cysts, posterior intra-thoracic goitres and parathyroid tumours.

The neurogenic tumours usually lie far back and in close relation to the necks of the ribs. Nerve lesions are common with tumours of this group, particularly those which are apical. Horner's syndrome is frequently present. Twenty per cent. of these tumours have a dumb-bell extension and erosion of bone is often seen.

The gastrogenic cysts (or reduplication cysts of the oesophagus) are most common in infants. They may be small but are often very large and may erode the ribs.

The posterior intra-thoracic goitre moves when the patient swallows and there may be absence of the thyroid in the neck on that side, or there may be an obvious connection with the cervical thyroid.

Bronchogenic cysts are characteristically found just below the bifurcation of the trachea and may move when the patient swallows. They may cause dysphagia and may be within the wall of the oesophagus.

Tumours of the lymph nodes—the lymphosarcoma group, the tuberculomata and

TABLE 1.

	Blades	Bradford <i>et alii</i>	Brewer <i>et alii</i>	TOTAL
<i>Benign Tumours.</i>				
Neurogenic tumours	29	6	16	51
Bronchogenic cysts	23	6	3	32
Teratomata and dermoid cysts	14	4	8	26
Pericardial cysts	10	7	2	19
Thymomata	4	2	2	8
Lipomata	3	4	0	7
Thyroid adenomata	2	1	2	5
Gastrogenic cysts	1	1	0	2
"Tuberculomata"	2	0	0	2
Leiomyomata	0	1	1	1
Fibroma	1			
Lymphangioma		1	1	
Boeck's sarcoid	1	5		5
	94	36	35	165
<i>Malignant tumours.</i>				
Teratomata	6	0	0	6
Thymomata	2	1	0	3
Neurosarcoma	1	0	1	2
Lymphoblastoma	2	0	0	2
Hodgkin's disease	4	1	3	8
Sarcoma			5	5
Carcinoma		3		3
	15	5	9	29

Boeck's sarcoid are found in association with lymph nodes and therefore along the course of the trachea on both sides and below the arch of the aorta on the left side.

The greatest difficulty in diagnosis probably is to distinguish the posterior apical tumours from each other. In this area neurogenic tumours, pleural exclusion cysts, superior sulcus tumours and even meningocoeles or aneurysms of the subclavian artery may occur. All of them may be associated with a Horner's syndrome, with other evidence of nerve pressure or with erosion of the ribs. Severe pain is in favour of a malignant growth as is irregular erosion of the ribs or extension into the soft tissues of the back.

In the inferior mediastinum neurogenic tumours, gastrogenic cysts and diaphragmatic herniae must be considered. The gastrogenic tumours lie posteriorly and X-rays combined with a barium swallow should disclose a diaphragmatic hernia. Further the hernia has air in it and is the only tumour in this area that has.

It should be sufficiently obvious from the data presented here that if a mediastinal tumour is encountered a certain diagnosis can only be made by excision of the tumour; that excision of the tumour is nearly always possible, and that if the tumour is symptomless almost certainly it will be removable but almost certainly if not removed it will produce symptoms in due course.

It is difficult to estimate the frequency with which the various types of tumour occur. In three recent large series of cases the incidence of the tumours has been influenced by mass surveys mainly of young adults and have not included cases garnered from hospitals for children. Nevertheless the figures are of interest. The series of Blades (1946) 109 cases, Brewer and Dolley (1949) 44 cases and Bradford, Mahon and Grow (1947) 41 cases have been combined. Thus there are 194 cases. Blades collected his cases in 3 years at an army centre. Gastrogenic cysts and ganglioneuroma which occur mainly in infants and children respectively are insufficiently represented in this collection (Table 1).

Table 2 shows the incidence in our series in which 75 per cent. of the patients sought advice for symptoms due to tumour.

In all series only cases referred for surgery are included.

TABLE 2.

Bronchogenic cyst — — — — —	0
Miscellaneous cysts — — — — —	3
Neurogenic tumour — — — — —	1
Teratomata — — — — —	4
Pericardial cyst — — — — —	1
Thymoma — — — — —	0
Thymoma of infants — — — — —	3
Lipoma — — — — —	1
Thyroid adenomata — — — — —	2
Oesophageal cyst — — — — —	0
Lymphangioma — — — — —	1
Osteochondroma — — — — —	2
Possible Cyndroma — — — — —	1
Aneurysms — — — — —	2
Haemangioma — — — — —	1
Malignant Tumours.	
Superior sulcus tumours — — — — —	2
Hodgkin's disease — — — — —	1
Cardiac tumour — — — — —	1

## TERATOMATA.

The teratomata and dermoid cysts of the mediastinum with their bizarre contents—hair, sebaceous material, teeth, bone, and other tissues—have long been recognized as one of the more common of the mediastinal swellings.

Rusby (1944), in a survey of 251 cases which included six of his own, states that the first recorded example was the subject of an address in 1823 in London by J. A. Gordon. The cyst was diagnosed at autopsy but symptoms due to its presence had been produced during life.

As these cysts are developmental in origin and as about 80 per cent. of the patients present with symptoms between the ages of

10 and 40 presumably the tumours do not cause symptoms until, in their growth, they exert pressure on contiguous structures. If they happen to arise amongst the vessels and near the base of the heart, pressure symptoms will be produced early, but if free to enlarge they may grow to colossal proportions before their very bulk causes dyspnoea. Thus they are among the largest of the mediastinal tumours.

One of the most interesting findings of Rusby was that although the cysts may be found incidentally at autopsy or dissection, of his 209 cases there were only 9 or 4.3 per cent. in which it was clear that the tumour did not contribute in any way to the cause of death. Of these 9 one patient was 37 years old, two 30 and one 25. Thus it is almost certain that less than 5 per cent. of these tumours will not cause symptoms at some time. Further he showed that the average time between the appearance of the first definite symptoms of the tumour and death was 1.8 years in 49 cases in whom there was no active treatment other than exploratory puncture. Too much regard should not be paid to such an average but nevertheless it emphasizes the need for treatment.

The pathology of these tumours has been the subject of hot debate and fantastic speculation. Nicholson has pointed out that many of the theories of teratoma have resulted from pre-conceived ideas rather than a study of morphology and embryology (Nicholson, 1950).

Willis (1948) believes that the teratomata are tumours arising from foci of plastic pleuro-potential embryonic tissue which escaped from the influence of the primary organizer during early embryonic development, this escape being in some way related to disturbances emanating from the invaginated organizing tissues of the primitive streak and so affecting median or paramedian parts in close relationship to these tissues. The affected primordium, as it grows, differentiates in accordance with its own intrinsic labile determinations, producing a variety of tissues foreign to the part in which it grows. If these tissues differentiate and mature as fast as they grow a benign cystic teratoma results. If the tissues

fail to mature completely but retain their capacity for continued growth at the embryonic level a malignant embryonic teratoma results.

These tumours were formerly divided into 3 groups, epidermoid cysts, dermoid cysts, and teratomata, but even if there was a pathological basis for such a division it has little practical importance. From the clinical point of view the tumour may contain bone or other radio-opaque material and will contain some sebaceous material or fat and probably some hair. The fat content may be liquid and often of varying density so that if the patient is kept in an erect position for some time fat may settle in such a way that a level can be seen on radiological examination.

The tumours may be entirely cystic or solid or solid with cystic areas. They may be irregular in shape and the irregularities are often angular rather than curved.

They are found in the anterior mediastinum but generally encroach upon one or other side of the midline and when very large may appear to have no connection with the mediastinum but careful inspection of radiographs or the specimens will show a pedicle extending to the midline; in 166 cases in which the site was mentioned Rusby found that 88 extended to the right side and 65 to the left and 13 were central.

They may occur within the pericardium and Willis (1948) has referred to five such cases. Gebauer (1943) gives an account of one attempt to remove such a tumour and Beck (1942) did in fact remove an intra-pericardial teratoma.

The incidence is about equal in the sexes.

The symptoms produced by teratomata are those due to excessive growth, those due to pressure, and those due to complications.

The size of the tumour may be such that the respiratory reserve is reduced with consequent dyspnoea. The tumour may present in the neck as in Gordon's case or high on the chest wall. The thorax may be enlarged on the side of the tumour and Harrington quotes a case in which the patient's tailor drew his attention to this.

The symptoms due to pressure are cough, often dry and irritating, both to the patient and to others. It may occur in spasms and terminate in haemoptysis. Dyspnoea on effort is common and there may be smothering spells.

Pain is frequent. It may be in the chest wall like that of pleurisy but more commonly is a dull ache. The pain may radiate down the arm into the fingers or into the face and ear. There may be abdominal pain.

Engorgement of the veins of the neck may be present and accompanying it some slowness of cerebration. This is apparent after removal of the tumour when the patient notices that his mind seems clearer.

Palpitation may occur. Evidence of pressure on the recurrent nerve or sympathetic chain may give rise to alterations of voice and Horner's syndrome respectively.

The pressure symptoms, as with other mediastinal swellings, may vary with changes of posture.

These tumours are subject to sundry complications. They may become infected apparently by blood borne organisms or the infection may be introduced from without during aspiration of the cyst. If the cyst is already very large the infection may so obscure the picture that the condition is regarded as an empyema as in Case 4; the diagnosis is then very difficult. The facts that the supposed empyema is in a most unusual situation and does not pursue a course consistent with an empyema, or that there are contents recognizable as those from a teratoma, sooner or later enable the correct diagnosis to be established.

Rupture of the cyst may result from pressure necrosis or from infection of the wall. Rupture has occurred into the pleural cavity, aorta or the pericardium, but is more usual into a bronchus with the establishment of a fistula into the cyst. Cough is a premonitory symptom and, after rupture, the contents of the cyst may be coughed up. At this stage there is copious sputum in which hair or sebaceous matter may be seen by the startled patient, who, no doubt in older and allegedly more barbarous times, would have had a difficult task to avoid a

charge of witchcraft. Not the least interesting point in regard to the hair of dermoids is that it is not always the same colour as that of the host! The formation of a fistula is usually followed by infection of the cyst with all the sequelae of persistent infection in the bronchial tree, such as cough with production of foul sputum and debris, haemoptysis, pyrexia, toxæmia and clubbed fingers; but occasionally the contents of the cyst are evacuated after some months and the fistula closes without infection. Prior to rupture the cyst becomes adherent to the lung or the organ involved and this suggests that infection is the probable cause. The adhesions increase the difficulties of surgical removal.

About 13 per cent. of the 209 tumours reported by Rusby (1944) became malignant. Included in this number is Lanza's (1937) case in which a secondary deposit from a malignant laryngeal growth was found in a simple dermoid cyst. Although sudden enlargement of the tumour might be expected to be the first evidence of malignancy, sometimes the earliest evidence is the appearance of metastatic deposits in lungs or elsewhere.

In diagnosis there are several special points related to teratomata. They may contain teeth or well formed bone in which case the diagnosis may be made with confidence. If calcified areas are seen dermoid cyst must be considered but aneurysms, neurogenic tumours, thymomata and goitres may also contain calcified areas.

An angular irregularity is a helpful point in the distinction from aneurysms in which the outline is usually curved.

The dermoids are anterior in position. A pedicle may be visible.

If the patient remains still for some time before radiological examination a fluid level, as described by Phemister (1936), may occasionally be seen. The Aschheim-Zondek test may be positive if there be active hormonal gland tissue in the tumour.

The treatment of these tumours is undoubtedly surgical removal. Sooner or later if the patient lives long enough all these tumours will cause trouble. They should therefore be removed when diagnosed.

Like all other tumours of the mediastinum they are now being found with increasing frequency as a result of mass radiological surveys.

If removed while the patient is still symptom-free, operation will almost certainly be easy and successful.

When the tumours are very large not only is the operation difficult but adjustment to the altered intra-thoracic pressure may be slow and sudden collapses are frequent. It is possible that this complication can be avoided by careful adjustment of pleural pressures in the immediate post-operative period.

We have had four teratomata.

#### Case 1.

The first, a youth of 22, was found by routine service X-ray examination. His tumour was small, on the right side and contained teeth and bone.

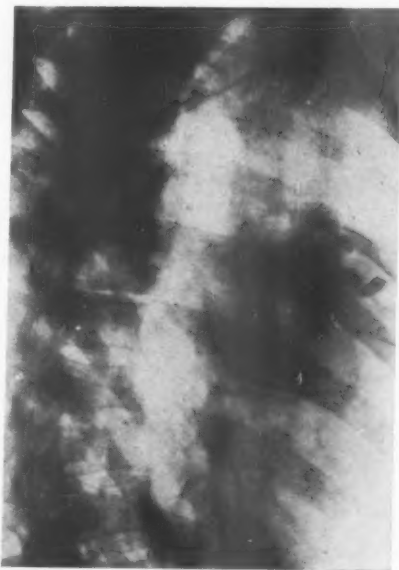


FIG. 1. Case 1. Right oblique X-ray. Teratoma. Note teeth and bone.

#### Case 2.

This case was also found on routine X-ray examination but afterwards he was found on cross examination to have some symptoms. This tumour was of considerable size and stretched from the

5th to 10th rib and from the mediastinum to the right axilla. It had the angular appearance mentioned earlier and it was situated anteriorly (Fig. II).

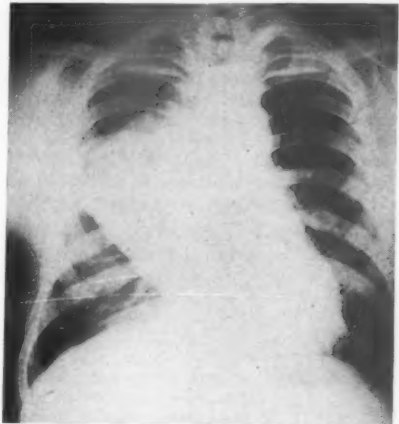


FIG. II. Case 2. Postero-anterior X-ray. Teratoma. Note angular outline.

#### Case 3.

A male, aged 40, was admitted on 30th April, 1947, with a history, of 8 years duration, of pain in his lower anterior part of the thorax which radiated to his right shoulder. This pain was associated with severe attacks of dyspnoea and orthopnoea. He had been admitted to hospital in 1939 and on radiological investigation had been found to have a very large tumour with irregular outline in the right side of his mediastinum and chest and anterior in position; the tumour appeared to pulsate. A diagnosis of teratoma was made and operation advised and preliminary pneumothorax induced. At this stage the patient refused operation. In 1940 he developed productive cough. The cough was severe and in 1942 he coughed up "hair and butter." This he continued to do for some five months and there were frequent haemoptyses. This episode followed "treatment to dissolve the cyst."

Since then he had had an irritating cough and yellow sputum but no more hair or blood. He had pain in his upper part of the thorax and a feeling as if he had a ball behind his chest wall at about the level of the third costal cartilage. The lower anterior part of the chest wall on the right side was tender.

On examination he was a thin man who looked older than his age. His face was dusky and slightly congested. His fingers were clubbed. His blood pressure was 100/60 mm. of mercury.

There was slight bronchophony in the right hilar region anteriorly. His heart and other systems appeared normal.

Radiological studies showed a shadow in the anterior mediastinum which extended out from the sternum in a wedge-shaped manner for about 1½ inches. Bronchograms demonstrated a pear-shaped dilatation of the pectoral branch of the right upper bronchus.

A diagnosis was made of teratoma of the mediastinum with residual bronchiectasis, and it was anticipated that a connection between the bronchus and the cyst would be found. Operation was accepted by the patient this time. While awaiting operation he had an episode of breathlessness with crepitations in the chest, right sided chest pain which radiated into the shoulder and he said his heart was missing beats.

Anaesthesia was pentothal, intra-tracheal nitrous oxide-oxygen and ether, and an anterior approach was made with resection of the 2nd rib. The right lung was adherent to the pleura but was easily stripped off the tumour, which was multi-locular about the size of two golf balls, a smaller cyst also being present. The larger cyst was lying between the vena cava and the aorta and firmly adherent to the aorta. It was separated by sharp dissection and the cysts were removed after evacuation of the content of the loculus adherent to the aorta. The left pleura was opened in the process of removal and closed by suture. The cavity was filled with penicillin solution and the chest closed with an under-water drain. Although there was no evidence of a fistula into the lung, and no cyst remnant could be palpated in the lung, the upper lobe was adherent and collapsed. The length of the operation and the patient's condition did not allow of lobectomy.

He made a good recovery. He lost his dusky appearance, his face was less congested and he volunteered the statement that his mind was clearer. His pain was relieved but he still had a cough.

For three years he refused further operative treatment for the residual cough and bronchiectasis. He then returned. At this operation a posterior approach was used. The upper lobe was everywhere adherent and there were very firm, almost fibrous adhesions to the old tumour site. The lobe was freed everywhere else and the vena cava was separated from the adherent lung. In the subsequent process of dissection of the adherent lung the vena cava was damaged at the level of the entry of the left innominate vein. The hole in the vein could not be secured by lateral ligature or suture and the vein was ligated just above the entry of the left innominate vein, though its calibre was greatly reduced. His face became very congested indeed and his condition deteriorated considerably, but, after a period of observation, the chest wall was closed. His colour improved, and the congestion diminished; within two hours his face appeared normal and he has had no further congestion. His cough was greatly improved and his sputum diminished. Apparently this was due to the fact that his upper lobe remains collapsed. He is undecided whether to have a further operation or not. He has returned to work.

The cysts contained sebaceous material and hair and the wall of the large one was calcified in places. Microscopic examination showed that the cyst wall was completely hyalinized with lipoid degeneration and calcification of inner aspect. All traces of original structure had disappeared.

#### Case 4.

The patient, a girl aged 15, was admitted on the 10th Oct., 1949. She had had rheumatic fever one year previously. In July, 1949, she had pneumonia and pleurisy but was discharged from hospital in September. Two weeks previous to admission she caught a cold. She had a slight cough and on admission a temperature of 100.5°.

Physical examination showed that her colour was good. Her blood pressure was 130/90 mm. of mercury. There was considerable right cardiac dullness and a systolic murmur was heard at the aortic area. Her chest was dull to percussion on the left side anteriorly as far as the axilla, with diminished breath sounds in this area. Neither liver nor spleen was palpable.

Radiological study indicated that there was a dense opacity of the left hemi-thorax. It occupied most of the chest and was anterior in position. There was normal lung posteriorly and at the apex. There was marked displacement of the heart to the right.

A diagnosis was made of encysted pleural effusion in the upper anterior two-thirds of the left chest. Her haemoglobin was 12.9 grms. per cent., 9,000 leucocytes of which 74 per cent. were neutrophils. Blood culture was negative. The Mantoux and Casoni reactions were negative.

On 19th Oct., 1949, the left chest was aspirated through the third intercostal space anteriorly. The needle passed through tough and thick tissue to a depth of 4½ inches and 400 cc. of brown turbid non-smelling pus was obtained. Penicillin (100,000 units) was injected.

Laboratory report was that the fluid was sterile on aerobic and anaerobic culture. There were many polymorphonuclear leucocytes present in a smear but no organisms.

There was no improvement after this and no change in radiological appearance and seven days later a further aspiration was attempted without success.

On 3rd Nov., 1949, the left chest was more prominent anteriorly and measured ½ inch more than the right chest at the 3rd rib. She had slight pyrexia up to 100°.

Next day a thoracotomy was made under pentothal anaesthesia. Portion of the 6th rib was resected in the anterior axillary line after pus had been aspirated there. Pus gushed out after the pleura was opened. An under-water drain was established.

At bacteriological examination of the fluid *Alcaligenes faecalis* grew on culture but organisms were not seen in the smear.

There was considerable drainage for several days but it then diminished and a pyrexia up to  $103^{\circ}$  of intermittent type developed. On 22nd Nov., 1949, the tube track was dilated and 24 ozs. of pus aspirated and drainage re-established. After this procedure the pyrexia subsided but there was no indication that the cavity was smaller. Although the lung could now be seen above the shadow the heart remained displaced to the right.



FIG. III. Case 4. Postero-anterior X-ray. Teratoma. Note displacement of the heart to the right, and bulge of the left chest wall.

Radiological examination after injection of lipoidal into the sinus showed an irregular cavity with branches below and posterior to a rounded anterior opacity which extended up to second costal cartilage.

A further exploration of the cavity was made on 24th Jan., 1950, and it was seen to be bounded posteriorly by lung and anteriorly by a thick wall. The cavity was irrigated after this operation and two days later there was evidence of a bronchial fistula into the cavity.

The position did not change substantially for some months when a bronchogram was made. This showed that the lung was displaced posteriorly by a large opaque area with a rounded posterior margin. The opaque area extended at least to the manubrium sterni. The empyema cavity formed the lower and posterior border of the area.

A diagnosis was now made of a thick walled tumour in the anterior mediastinum with an empyema outside it.

On 13th June, 1950, a further operation was performed under pentothal, curare and cyclopropane anaesthesia with controlled respiration.

An anterior approach was made and portion of the 6th rib was resected.

There was some pus in the empyema cavity and a thick walled cystic tumour was seen. It was anterior in position and extended from the diaphragm up to the neck. The tumour was adherent to the pericardium. A plane of cleavage was found and the tumour freed easily, except from the pericardium, portion of which was removed with the tumour, until the upper pole was reached. There appeared to be a pedicle here which extended high and medially into the mediastinum. During the dissection of the pedicle trunk venous haemorrhage was encountered. It appeared to be coming from the subclavian vein which was tied without effect. As the patient's general condition was poor at this stage the bleeding was controlled by packs which were left in place. The tumour was removed except for a small portion in the region of the haemorrhage. The chest was closed with an under-water drain. The post-operative course was stormy. The patient was drowsy and there was some intestinal ileus. She had considerable pyrexia and on 20th June with a similar anaesthetic the chest was re-opened and the packs removed. The haemorrhage recurred at once from an area near the left auricle. Several attempts to secure the bleeding point failed and further packs were inserted and the chest closed as before.



FIG. IV. Case 4. Postero-anterior X-ray, with empyema cavity outlined by Lipiodol. The filling defect caused by the tumour is well defined.

She remained very ill and died 3 days later. The tumour was a teratoma.

Microscopical examination disclosed a teratoma of multiple adult tissue. No evidence of malignancy was found (Figs. III, IV, and V).

Apparently in this case the first indication of the presence of the teratoma was infection of the compressed lung. This settled down but the patient was not well, probably because portion of the tumour was infected. The tumour was aspirated and she then developed an empyema around the tumour. This obscured the picture and the diagnosis was not established until the bronchogram showed that the position of the lung precluded a diagnosis of interlobar empyema.

This case illustrates the difficulties which arise from infection, from a large bulky tumour, and from a pedicle which extends into the anterior portion of the superior mediastinum.



FIG. V. Case 4. Teratoma. Photograph of specimen.

#### MEDIASTINAL CYSTS OF DEVELOPMENTAL ORIGIN.

Until the end of the 1939-45 war reports of mediastinal cysts of developmental origin were but few. Since then three large series of mediastinal tumours have been published by Blades (1946) 109 cases, Bradford *et alii* (1947) 41 cases, and Brewer and Dolley (1949) 44 cases, a total of 194 cases. These series included material available as a result of the various routine surveys begun and carried on as an indirect result of the war. Thus many of the cases reported were symptom free, whereas in cases previously reported, apart from a few found at autopsy, in almost all, the cysts had caused symptoms.

Of these 194 tumours no less than 53 were cysts and we have been able to find case reports of 126 others which include 4 now reported.

It is possible to separate from this material four well defined groups of cysts of which the origin is reasonably certain. There is also a group of miscellaneous cysts of doubtful origin. This group includes those in which there is insufficient data for adequate classification.

#### Bronchogenic Cysts.

The first group is characterized by the presence in the cyst wall of structures commonly seen in the respiratory tract. They are lined by an epithelium which may be columnar, cuboidal or pseudostratified but is always ciliated. There may be cartilage, smooth muscle, or mucous or serous glands in the wall.

These are the bronchogenic cysts. They are found in close relation to the bifurcation of the trachea usually below or caudad to it and anterior to the oesophagus but they may extend more to one side or the other and be in relation to the wall of the oesophagus. There is often a pedicle attached to the carina of the trachea. The carina may be widened. They may be found included within the wall of the oesophagus—the intramural oesophageal cysts, but even these often have a pedicle attached to the carina.

Laipply in 1945 was able to find only 35 of these cysts. Since then we have been able to find 13 in the literature and there were 32 in the material of Blades, Bradford and Brewer.

We have been able to find 26 case reports with full details in addition to 26 examples with but few details.

Of the 26 cases, in 19 the cysts were closely related to the bifurcation of the trachea and 4 of them were within the wall of the oesophagus. Four others were in the posterior mediastinum between the hilum of the lung and the diaphragm. One was in the posterior mediastinum, and one anterior.

As these cysts in most instances are closely related to the trachea they may move when the patient swallows. Transmitted pulsation has been observed.

Fifteen of the patients were between the ages of 20 and 40; the youngest patient was 5 months old and there were two over 60.

Sixteen were males.

Twenty-nine of the 52 patients were symptom free.

Pain was the commonest symptom and cough and dyspnoea were also noted. Dysphagia was present in several cases though absent in one of the intra-mural cysts of the oesophagus. There may be a productive cough if there be infection in collapsed lung, or if there be a fistula between the cyst and a bronchus (Hardy, 1949, Robb, 1950).

The treatment of these cysts is complete excision. Partial excision has been done several times and is an unsatisfactory procedure.

The contents of the cysts are often mucoid but may be purulent or blood-stained.

It is possible that infection of the cysts is responsible for the development of symptoms; in some cases the cysts have been known to be present for years without causing symptoms.

#### *Gastrogenic Cysts.*

The second group are the gastrogenic cysts also called reduplication cysts of the oesophagus or enterogenous cysts. The characteristic of these cysts is that portion or all of them is lined by gastric mucosa. In the best developed the mucosa is in rugae and easily recognized as gastric. In others the gastric mucosa occurs only in patches. The rest of the cyst may have respiratory ciliated epithelium, duodenal, or intestinal epithelium. Outside the mucosa there are the two layers of muscle characteristic of alimentary canal. The gastric mucosa may only be found after a prolonged search and it is probable that cysts believed to contain intestinal mucosa only do, in fact, have some gastric mucosa as well.

In many of the cases reported there has been evidence of activity of the gastric mucosa and in a case reported by Seydl (1938) a peptic ulcer developed and perforated into the pleural cavity and a bronchus.

The cysts may be small and rounded or may be large and extend the full length of the thorax. They are characteristically found in the paravertebral gutter in close relation to the oesophagus to which they are often intimately bound.

In 17 of these cysts all were posterior; 11 were on the right side and four on the left. Two were stated to be in the posterior mediastinum.

Twelve were in infants under 2 years old and mostly under one year; the oldest was in a 27 year old. Eleven were males and in 9 cases there was evidence of activity of the acid secreting mucosa of the cyst.

From the early age of the patients in whom these cysts have occurred it seems likely that the mucosa must sooner or later become active in most of them and lead to death. The age incidence is in striking contrast to that of the bronchogenic cysts. Blades found only one of these cysts and Brewer none which suggests that they cause symptoms or death at an early age.

Pressure symptoms are common and respiratory embarrassment frequent when the cysts are large. There may be infection in collapsed lung with productive cough, dyspnoea, haemoptysis and clubbed fingers. There were only two patients without symptoms.

Radiologically these cysts lie posteriorly. In 3 of the cases there was erosion of vertebrae or ribs, presumably from pressure.

Treatment should be complete excision of the cyst. If there is dangerous respiratory embarrassment some measure short of this may be required, such as marsupialization and drainage with subsequent removal. In such cases almost invariably there has been excoriation of the skin from the acid secretion of the cyst.

The cysts are usually closely bound to the oesophagus and it is important that in removal of the cyst, the oesophagus should not be narrowed if a portion of its wall has to be excised.

The contents of these cysts are viscid mucoid fluid which may be yellow or blood stained and of an acid reaction.

#### *Pericardial Coelomic Cysts.*

The third group of cysts are those characterized by clear contents and by an epithelium of flattened cells resembling endothelium. It is these cysts which have been called spring water cysts.

We have found 72 such cysts and there are case reports of 35. Thirty-nine of the 72 patients were symptom free, and the cysts were found by routine survey.

The cysts are found in close relation to the pericardium but usually are not attached to it. However, there may be a stalk-like attachment and in some cases a communication with the pericardial cavity is present in the stalk. This opening may be narrow or broad, and these cysts are then properly pericardial diverticula. Of 46 cases, there were 9 diverticula and 7 with a stalk.

The cysts may be multiple and they occur most frequently at the anterior pericardio-phrenic angles and on the right side, and 24 of 35 cases were at this site and six were on the left; four were in front of the pericardium at the base of the heart, and in one the position was not stated.



FIG. VI. Case 5. Lateral X-ray of cysts of the mediastinum.

Pain in the chest, a sense of tightness in the epigastrium or of pressure in the chest and tachycardia were seen in these patients who had symptoms. The symptoms were often relieved by alterations in the posture of the patient.

None of the patients with pericardial cysts was under 27 years of age. Seventeen of them were under 50.

#### Case 5.

A female of 23 months had pink disease at the age of 1 month; since then she had productive cough with occasional haemoptysis. On examination there was some finger clubbing, and there was bronchial breathing with numerous crepitations on both sides of the chest. Radiological studies showed evidence of bronchiectasis and consolidation at both bases with an anterior mediastinal shadow extending up to the aorta. She had several bronchoscopic examinations but developed meningitis and died after 2 months in hospital.

Post-mortem examination showed advanced bronchiectasis with peri-bronchial inflammation in both lower lobes of the lungs and pneumococcal meningitis.



FIG. VII. Case 5. Cysts of the mediastinum bronchogenic. Photograph of the posterior aspect of the anterior chest wall. The xiphisternum is below. The chest wall has been mounted on the lungs which are visible behind. Note the small left-sided cyst with a piece removed; the larger cyst with veins over its surface, and the medium sized cyst above it.

Three multi-loculated thin-walled cysts were present attached only to pleura. The largest, measuring  $3\frac{1}{2}$  inches by 2 inches by 2 inches, occupied the anterior portion of the right side of the thorax, being attached over the region of the third, fourth and fifth costal cartilages. A similar but smaller cyst, measuring  $1\frac{1}{2}$  inches by 1 inch by  $\frac{1}{2}$  inch, occupied a symmetrical position on the left side. The contents of these two cysts was a clear faintly opalescent fluid. The third cyst was about  $\frac{3}{4}$  inch in diameter, spherical in shape and attached at the cardio-phrenic angle on the right side and contained blood stained fluid (Figs. VI, VII and VIII). Histological examination of one of the cysts showed it to have a wall composed of fibrous tissue with many vessels and occasional strands of smooth muscle possibly derived from the vessel walls. There was no epithelium visible and there appeared to have been inflammation of the cyst wall. In the lumen there were many cells of a signet ring shape suggesting mucin-containing macrophages.

These cysts were multiple, one was at the cardio-phrenic angle and two of them contained clear fluid; however, the wall appears to have been thicker than in the pericardial cysts. Further no epithelium was available for identification in any of three slides, but the presence of the signet ring cells suggests that the cysts may have contained some mucin. They may then be bronchogenic cysts in an unusual situation or they may be peripheral lung cysts which have become sequestered from the lung.

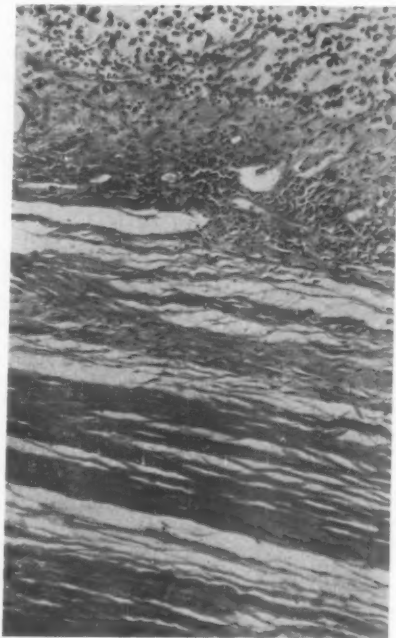


FIG. VIII. Case 5. Cyst of the mediastinum. Low power ( $\times 90$ ). Photomicrograph of the cyst wall with granulation tissue in the lumen. The laminated structure of the wall is well displayed.

In the fourth group of cysts there are some which are lined by a flattened or cuboidal epithelium, and have a wall of laminated fibrous tissue, and are found at the apex of the thorax and posteriorly. Two such cases are reported here.

#### Case 6.

A male, aged 49, for five years had had pains in right shoulder, right arm and right side of face and chest. On examination he was a spare individual; his right pupil was smaller than the left and there was anhydrosis on the right side of the face. There was no wasting of the small muscles of the right hand. The Wassermann and Casoni reactions

were negative. Radiological study showed an opacity filling the apex of the right chest, posterior in position, with a clear cut rounded lower border.

At operation on 4th June, 1940, through a posterior approach, an extra-pleural cyst was found lightly adherent to the sides of the third and fourth thoracic vertebrae; it was easily removed. It contained two loculi in one of which there was brownish necrotic material with a foul odour. The fluid on culture grew gram positive bacilli and cocci. In the other there was laminated blood clot. On microscopic examination the wall of the cyst was composed of laminated fibrous tissue with some young granulation tissue. No characteristic epithelial layer was seen.



FIG. IX. Case 6. Pleural sequestration cyst. Photomicrograph. Low power ( $\times 90$ ). Note the laminated wall and structureless lining.

His post-operative course was uneventful. His "neuritis" was relieved by the operation but his pupil remained unchanged. He was still free from symptoms eighteen months later (Fig. IX).

#### Case 7.

A male, aged 63, was admitted on 25th July, 1949, because of a recent haemoptysis. He gave a history of slightly productive cough for years and frequent colds. He had had a haemoptysis four years previously and dyspnoea on exertion for two to three years.

Physical examination revealed an elderly man with unequal pupils, the left being larger than the right. The heart and lungs were normal; blood pressure was 140/85 mm. of mercury. There was no wasting of the small muscles of the right hand. The Wassermann reaction was negative, and there were no acid-fast organisms in the sputum.

Radiological survey showed an apical and posterior shadow in the right side of the thorax with a rounded lower margin about level with the

anterior end of the first rib. There was no evidence of enlargement of the intervertebral foramina, or of erosion of ribs. There was no evidence of tuberculosis. Bronchogram showed the tumour to be extra-pulmonary.

A diagnosis of extra-pleural cyst or ganglioma was made.

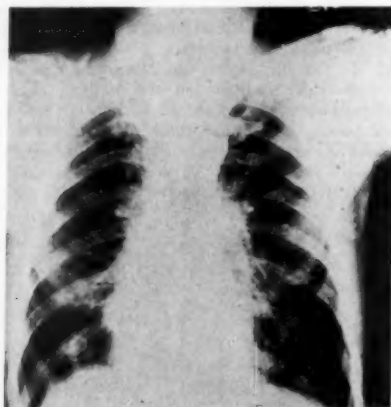


FIG. X. Case 7. Postero-anterior X-ray. Pleural sequestration cyst at the right apex.

At operation on 25th Aug., 1949, by a posterior approach an extra-pleural cyst was found and easily enucleated. It measured 3 inches by 2 inches by 2 inches and was lying alongside the bodies of the second and third thoracic vertebrae with an indefinite attachment to the sides of these bodies.



FIG. XI. Case 7. Lateral X-ray. Pleural sequestration cyst. Note the rounded border.

Macroscopically the cyst had a wall about 3 mm. thick with yellow serous content which soon clotted. The wall was smooth but there were some small raised areas which made the thickness of the wall 5 mm.

Laboratory report on the fluid was that it was blood clot only.

Microscopic examination showed a similar picture to that seen in Case 6. The same laminated fibrous tissue wall was present. There was some squamous epithelium up to 2 cells thick. Cilia were not seen neither were cartilage nor glands (Figs. X, XI and XII).

His post-operative course was uneventful.



FIG. XII. Case 7. Pleural sequestration cyst. Photomicrograph. High power (x400). Note similarity to Case 6. The lining here is of round cells but in other areas was a squamous epithelium about 2 cells deep.

In both these patients there was disturbance of the cervical sympathetic nerves on the same side. In both there was a posterior and apical cyst. The histological picture was similar and differs from that seen in the cysts already described. Case 10 of Brown and Robbins (1944) would seem to be in almost the same anatomical situation but the details are insufficient to classify this case.

In addition to the cysts in this fourth group are others, with a similar histological picture, found along the mediastinum

(d'Abreu, 1937). It is probable that they are pleural sequestration cysts characterized by a wall of laminated fibrous tissue with scanty nuclei and lined by a single or double layer of flat or low cuboidal cells. These cysts appear blue in colour and their walls are opaque.

There remains, however, several cysts of doubtful origin. Some of these have similar fibrous walls but the lining may be stratified, squamous, columnar or cuboidal epithelium (Greenfield, 1943). In some cysts no epithelium may be found as in Case 6. It has been thought that the lining epithelium in some of these cysts has atrophied and disappeared as a result of pressure or of infection. Nevertheless, many of the cysts in the first three groups were infected and yet the characteristic epithelium could be seen. It may be that the cysts in which the epithelium is stratified, cuboidal or columnar are of bronchogenic origin in which cilia and the other characteristic structures usually seen have failed to develop. It seems impossible at present to classify all the cysts into the four main groups described here. Possibly some of these in which no epithelium is found are old haematomata. This supposition is supported by the occasional presence of areas of calcification in the walls (Wright, 1936). In several cases insufficient details are available for classification and one such case is now reported.

#### Case 8.

A male, aged 67, died of acute pancreatitis. At post-mortem examination a cyst 4 inches in diameter was found in the left posterior mediastinum. It extended from the bifurcation of the trachea to the diaphragm and, although its epithelial wall lay closely across the bronchi and the oesophagus, there was no direct connection with these structures. The cyst was plum-coloured and distended with dark brown fluid. The benzidine test for blood in the fluid was positive. No cholesterol was detected in the fluid. Unfortunately no section of the wall is available.

From their position four of these cysts may be bronchogenic in origin but there is not enough evidence for diagnosis.

All these cysts appear to be developmental in origin.

The trachea develops from the pharyngo-tracheal ridge in the floor of the foregut, the rest of which forms the oesophagus.

The tracheal bud elongates and gives off the bronchial buds which develop into the lungs, all lined by respiratory epithelium.

It may be assumed that at one stage all this tissue which produces trachea, lung, oesophagus and stomach is competent to react to the appropriate stimulus and thus produce the right organ.

If the evocator's sphere of influence is wider than it should be then tissue will be induced to form the required tissue but its organization may be incomplete and it seems that most of the anomalies can be explained along these lines; thus the bronchogenic cysts are produced by an evocation strong enough to cause a bud of tissue beyond its proper sphere of influence but not strong enough to lead to its full organization into lung; the result is a piece of tissue with most of the constituents of the bronchial buds but not the proper organization; similarly if the moulding of the oesophagus is faulty a piece of tissue is produced which is induced to form oesophagus and stomach-like tissue but is not properly organized. From the fact that most of the cysts of alimentary origin appear to have gastric mucosa in them at some place, stomach evocation appears to be the one at fault in these, but the influence of the lung evocation is sufficiently strong in places to produce some respiratory epithelium.

This would also suggest an explanation of Gans' (1951) case in which a lobe of lung tissue was found with a bronchus opening into the oesophagus. Apparently a bud was formed from the oesophageal portion of the tube at the hilar level but in the lung sphere of influence and a lung was produced.

The strictures and fistulae between oesophagus and trachea are determined by incomplete organization of the development of the trachea from the oesophagus as are the intramural cysts of the oesophagus which are lined by ciliated epithelium.

Lillie, McDonald and Clagett (1950) were impressed by the similarity of the pericardial cysts to the diverticula of the pericardium and as a diverticulum-like structure occurred during development they believe that they can explain these cysts and diverticula on the basis of a persistence of the ventral coelomic recess. If the recess persists

intact a diverticulum with a wide base results. If the proximal portion is constricted a diverticulum with narrow base results and, if completely pinched off, a cyst in the pericardio-phrenic angle is produced. If the recess is completely pinched off and left cephalad as the septum transversum moves caudally then those mesothelial cysts, which are found higher in the mediastinum than the pericardio-phrenic angle, result.

As to the cysts at the apex of the thorax such as those in Cases 5 and 6, the fibrous laminated wall suggests that these originate from the pleura. Such epithelium as is present is consistent with this view. It is possible that these cysts with laminated fibrous walls and low cuboidal or flattened squamous epithelium develop from portions of the primitive pleura which have become sequestered and pinched off from the pleural cavity.

#### *Cysts of Accessory Lobes of the Lung.*

Accessory lobes of the lung with short bronchi may cause difficulties in diagnosis if they become cystic, as the cyst may then be seen only in relation to the mediastinum. If infected they produce symptoms similar to those of lung abscess.

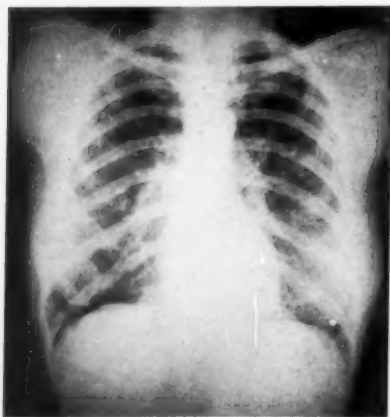


FIG. XIII. Case 9. Postero-anterior X-ray. Cyst of accessory lobe of the right lung. The edge of the cyst is just visible in the third right intercostal space.

#### *Case 9.*

A female, aged 40, for 10 years had had recurrent attacks of pyrexia and cough with production of much purulent sputum and occasional haemoptysis.

After a few days the pyrexia would subside but she would be left with a residual cough and up to 3 ounces of purulent sputum.

On examination she looked well but thin. There were no abnormal physical signs beyond an occasional rhonchus in the chest.

Radiological study showed a cavity, the edge of which just projected to the right of the mediastinum below the right lower lobe bronchus. Lateral projections showed it to be posterior to the right lower lobe bronchus. There was a fluid level in the cavity.



FIG. XIV. Case 9. Right oblique bronchogram. Cyst of the accessory lobe of the lung. A fluid level is visible in the cyst between the spine and the trachea.

Contrast studies demonstrated that it was to the right of the oesophagus and that two-thirds of the cavity was anterior to the oesophagus. Bronchograms gave no additional information. No lipiodol entered the cavity.

At the time of observation she was sputum free. On bronchoscopic examination there was a reddened area, with a little pus in its vicinity, on the posterior wall of the right lower lobe bronchus near the apical bronchus of the lower lobe. The bronchial tree was otherwise normal.

The site of the lesion seemed very unusual for a lung abscess but with this diagnosis in mind thoracotomy was undertaken.

An accessory lobe of the right lung was found. It originated from the right lower lobe bronchus below the apical segment. The lobe was posterior in position and lay against the mediastinum; it was quite free. There was an indentation in the medial and posterior border of the lower lobe of the lung below the apical segment. The lobe, in outline,

was shaped like a tennis racquet with the broad portion attached to the lower lobe bronchus. This portion was cystic. The handle of the racquet was fleshy atelectatic lung. The accessory lobe was excised and the bronchus closed.

Post-operative course was uneventful and she remains well nine months later, free from cough and sputum (Figs. XIII and XIV).

The cases on which this survey is based were found in the sources already given in the text and in the papers of Adams *et alii* (1943), Allison (1947), Carlson, (1943), Drash (1950), Freedlander (1939), Gledhill (1950), Kissner (1950), Ladd *et alii* (1944), Lambert (1940), Steele *et alii* (1945), and Swartz (1942).

#### TUMOURS OF BONY CAGE.

Chondromata or chondrosarcomata which arise from the posterior ribs or from the sternum may be very large indeed. They are irregularly calcified and as they enlarge their shadows overlap the mediastinum.

We have had two such cases.



FIG. XV. Case 10. Postero-anterior X-ray. Osteochondroma of the chest wall. The tumour extends across the midline within the chest.

#### Case 10.

A male, aged 75, employed (still!) as a gas-meter inspector, had a week's history of dyspnoea which had forced him to cease work. He had been unable to sleep and complained of pain in his

right chest. A companion noticed that he had a lump on the back of his right chest under the scapula and near the midline.

On examination his face was slightly congested when he was lying down but not when he stood up. There was a hard swelling about the level of the 4th to 7th ribs posteriorly which extended to the midline and under the right scapula which, however, moved freely over the surface of the tumour.

Radiological study by antero-posterior and lateral stereoscopic projections showed that there was a large tumour irregularly calcified which occupied the posterior region of the mediastinum and the right chest. It appeared to arise in the 6th rib. There was a second tumour related to the anterior end of the 7th rib (Figs. XV and XVI).



FIG. XVI. Case 10. Lateral X-ray. Osteochondroma of the chest wall encroaching on the mediastinum.

He was unaware as to how long the lump had been present. He found that he could sleep well with several pillows and felt so much better as a result that he resumed work as a trial. He does not desire any further treatment at present.

#### Case 11.

The second case was a man aged 63 who had had a lump in his epigastrium for many years. He developed pneumonia and a left lower lobe lung abscess from which he died soon after he was seen. The mass in his epigastrium was rounded, stony hard and had not moved with respiration. It had not been tender.

Radiological study had showed this epigastric mass to be portion of a very large irregularly calcified mass which extended up into the chest and mediastinum and apparently took its origin from the lower aspect of the sternum (Fig. XVII).



FIG. XVII. Case 11. Osteochondroma of the sternum.

The very large size of these tumours is characteristic as is the irregular calcification. The long history in the second case suggests that it was a benign osteochondroma. The radiological appearance is similar to that in Harper's case (1942).

O'Neal and Ackermann (1951) summarized a series of 96 such cases of osteochondromata of ribs and sternum of which 18 were related to the sternum or mediastinum. They point out, as have others, that most of these tumours are either malignant or become malignant and they should therefore be removed when diagnosed. Of the 18, 8 were reported as benign. The average age of onset was 34 years for the malignant and 38 years for the benign form and the sexes were approximately equally represented. The length of history in both groups

varied from a few months to many years. Six of the 11 malignant cases complained first of tumour and 5 of pain and in one case, in which the tumour arose at the thoracic inlet, Horner's syndrome as well. Six out of 7 of the benign cases complained of tumour, only one of pain.

There does not seem to be any criterion by which a certain diagnosis of malignancy or otherwise can be made by radiological examination.

The tumours, as in Case 9, may be multiple. If they are well calcified there is no difficulty in making a diagnosis of chondroma or chondrosarcoma; but in tumours, only slightly calcified, neurogenic tumours may cause confusion.

The tumours often undergo cystic degeneration. They are usually encapsulated to some extent but nodules of tumour may be adherent to surrounding tissues and thus be left behind if enucleation of the tumour is attempted.

The diagnosis of malignancy is made by the microscopic appearance of the tumour cells. Those of the malignant tumours vary in size and form and exhibit atypical nuclei of various degrees.

The treatment is wide excision of the tumour, the bones from which it arises, and, where this is possible, the tissues between the bones and around the tumour.

In cases of tumour which involve the mediastinum a combination of radical removal and enucleation may have to be attempted.

Where there are multiple tumours operation is only advised if there is evidence of increase in size.

Malignant cases have a poor chance of radical cure by operation and survive less than five years. The benign or malignant cases of low grade, with radical operation, should have a good chance of cure and of survival over five years.

(The second, and concluding, portion of this article will be published in the November issue of the Journal.)

## THE ADENOLYMPHOMATA.

By IAN HEINZ.

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**A**DENOLYMPHOMATA are tumours composed of epithelium and lymphoid tissue which usually arise in relation to the salivary glands. They are uncommon and comprise about 6 per cent. of all parotid gland growths. In this paper will be described 12 cases situated near the parotid gland and one which was removed from the false vocal cord.

### CLINICAL ASPECTS.

Almost invariably these tumours arise during the fifth, sixth and seventh decades although the extremes in previously recorded cases was a child aged two and a half and a man aged ninety-two years. In the present series of 12 cases the average age was sixty years and 9 of the tumours occurred in males.

The usual history has been of a slowly growing lump present for many months, not accompanied by pain or tenderness. In 9 cases the tumour was situated just beneath the angle of the jaw whilst 3 others were in the pre-auricular area. In the literature adenolymphomata have also been described as arising along the ramus of the mandible, near the submaxillary gland, and in the retro-auricular region. No instance of bilateral adenolymphomata was encountered although these have been described and there was no significant difference in the incidence on right and left sides.

The lesions present as soft, often fluctuant, smooth or lobulated swellings from 2 to 10 cms. in diameter which are not attached to the skin or deeper structures.

Their physical characters differ therefore from the commoner parotid adenomata and carcinomata. None of the cases was correctly diagnosed before operation. The tumours in the pre-auricular region along with most of those below the angle of the mandible, were recognized as parotid growths. In 3 cases, however, the swellings were thought to be enlarged lymph nodes and

this impression was strengthened at operation when the parotid gland itself was not visualized.

### PATHOLOGY.

Macroscopically adenolymphomata are well encapsulated, smooth or lobulated masses and in this series varied from 2 to 3.5 cms. in diameter. They are rounded or oval in shape and usually of soft consistency. The tumour cuts with ease and the surface thus displayed is pinkish-grey in colour but may vary in texture. In some adenolymphomata the surface is cystic in nature, in others firm and fleshy whilst many present an apparently friable and papillary structure which, however, does not break away on cutting. This latter appearance often enables a diagnosis to be made macroscopically. After the specimen has been in formalin any spaces present on the cut surface are seen to be filled with a greyish coagulum. The variation in the appearance of the surface is simply due to the degree of dilatation of the glandular structures and the presence or absence of papillary ingrowths into them.

### *Histological Appearances.*

The typical adenolymphoma has a very characteristic histological pattern which enables it to be readily recognized. The stroma consists of a fine reticulum containing lymphocytes and well-developed lymphoid follicles and it does not appear to differ in any respect from ordinary lymphoid tissue. On this background the epithelial cells are arranged in glandular or cystic pattern usually complicated by papillary ingrowths into the larger cysts. A low power view of such an area can be seen in Fig 1. There is a layer of tall columnar cells next to the lumen and these have an eosinophilic cytoplasm with the nuclei placed towards the free surface. Beneath them are smaller cuboidal basal cells resting on a thin basement membrane which is well defined in most areas. The epithelium has thus been described as

being of pseudo-stratified type. Fig. II is a photomicrograph illustrating the typical epithelium.

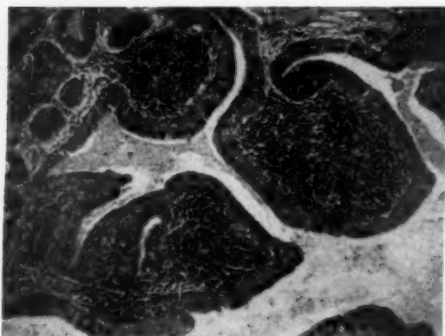


FIG. I. Photomicrograph of an adenolymphoma showing a cystic space with papillary ingrowths. There is a well developed lymphoid stroma. (x 180.)

The cysts contain mucoid material, desquamated epithelial cells and debris but cilia, which have been described, could not be demonstrated in any of the material examined.

The regular nature of the epithelial structures, the absence of microscopic invasion of the well-developed fibrous capsule and the infrequency of mitotic figures leaves no doubt as to the benign nature of a typical example.

#### *Variations in Histological Form.*

Lloyd (1946) has described 7 cases of a solid variety of "adenolymphoma" and thinks it fair to include them in the adenolymphoma group. This solid type of adenolymphoma, in which glandular structures can be seen throughout an otherwise normal lymph node, is depicted in Fig. III. The tumour which was removed from the left parotid gland of a middle aged female was of flattened pyriform shape, measured 4 x 2.8 x 1.5 cms. and presented a smooth, brown surface. The cream-coloured opaque tissue comprising the body of the growth was of firm consistency and uniform texture.

Yet another type of tumour with a lymphoid stroma containing Hassall's corpuscles is shown in Fig. IV. The cystic spaces and papillary projections all present a stratified squamous epithelium and this growth was rightly regarded as a branchial cyst. In

many respects it is similar to the adenolymphoma and in fact, these latter do on occasions contain squamous epithelium.

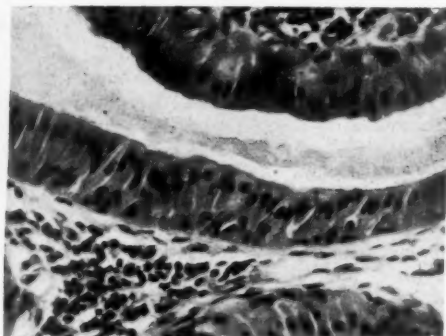


FIG. II. Photomicrograph showing the pseudo-stratified epithelium in greater detail. (x 350.)

Lloyd gives reasons for considering all forms of salivary adenoma as being closely related. This seems a wise attitude as many of the problems of the adenolymphoma group are artificial and due to a rigid adherence to definitely circumscribed examples. A gradation is seen between the typical adenolymphoma up to the more common "mixed" salivary tumour. There is the squamoid type, the epithelial type without lymphoid tissue and also those with a predominantly lymphoid structure.

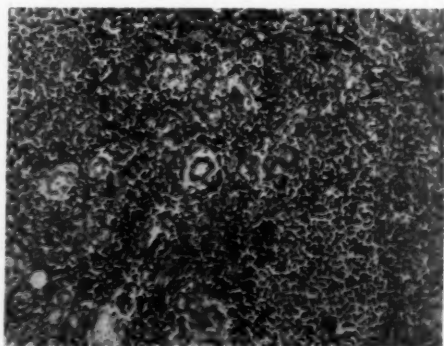


FIG. III. Photomicrograph of a solid type of parotid adenoma in which numerous glandular structures can be seen in an otherwise normal lymph node. (x 180.)

The sharp segregation of the adenolymphoma with columnar epithelium from the tumours in which the epithelium is squamous or squamoid in type is a good example of

a rigidity and artificiality of viewpoint which does not help in the comprehension of these growths nor assist in understanding the changes taking place in the tissues.

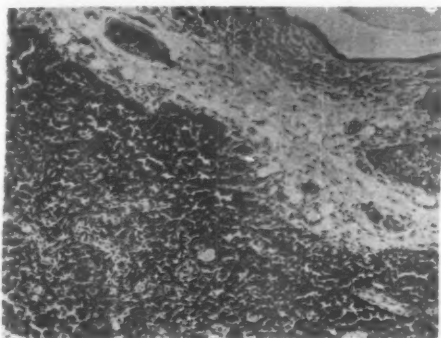


FIG. IV. Photomicrograph of a lateral lympho-epithelial cyst of the neck. Two Hassall's Corpuscles can be seen in the lower mid zone of the lymphoid stroma. (x 180.)

However, from the practical aspect there is much to be said for reserving the term adenolymphoma for the typical cases. This is because the group can be given an excellent prognosis. Admittedly four malignant adenolymphomata have been reported in the literature but these were atypical and contained squamous cells. There is nevertheless no basic difficulty; if the whole group were classified as adenolymphomata and qualified benign or malignant according to the recognized criteria no confusion could arise. The term would obviously only include those tumours in which lymphoid tissue was a prominent feature and would exclude those not containing it. The same problem is encountered in the segregation of lymphadenoid goitre from nodular goitres with marked lymphocytic aggregations.

#### *Pathogenesis.*

Adenolymphomata were first described by Albrecht and Arzt (1910). In their discussions of two cases they considered the tumour to be of endodermal origin and a result of tissue displacement.

In the subsequent years no fewer than eleven hypotheses have been advanced to explain the genesis of these growths. Many of the theories are very ingenious in that they attempt to show that certain embryological structures could be sequestered to

form the adenolymphomata but only three seem to warrant consideration. The view that they are of branchial origin has been widely held since the first examples were described and this was thought to be supported by the fact that in the branchial region lymphoid tissue and epithelium are in close association. Kraissl and Stout (1933) have, however, rejected this hypothesis after sound anatomical argument and King (1949) puts forward many cogent reasons for not accepting this same derivation for the so-called "branchial" cysts. Secondly adenolymphomata have been attributed to an abnormal blending of salivary and lymphoid tissue. Nicholson (1922) favours this theory of heterotopic salivary tissue in lymph nodes and quotes the work of Nisse. This last author in 1898 demonstrated salivary gland tissue in pre-parotid lymph nodes and the phenomenon has since been found to occur frequently. Fig. V is a photomicrograph showing salivary type epithelium in a pre-parotid node removed from a foetus. The possibility of such an association persisting late into adult life to form the adenolymphoma cannot be denied but such a hypothesis is really not necessary.

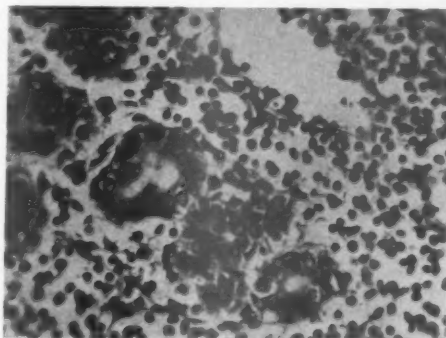


FIG. V. Photomicrograph showing glandular epithelium in a foetal parotid lymph node. (x 350.)

The third conception is of a lympho-epithelial relationship which is a fundamental feature of some tissues particularly in the neck and mediastinum. To explain this it is not necessary that tissue displacements should occur nor that epithelium and lymphoid tissue of adult form should become mixed mechanically.

The two tissues grow together either by one forming the other or by inducing its development in the adjacent tissues. The epithelium may (and usually does) demonstrably arise in the epithelium of a salivary gland or the glands of a mucous membrane. Other epithelial tumours, however, quite clearly arise in lymph nodes and appear to come from cells which have epithelial potentialities not apparent in the usual morphological form of epithelium.

The epithelial cells of an adenolymphoma may thus arise by metaplasia from epithelial ducts of glands or from cells which have been morphologically "endothelial."

The following case is of interest in that it has the typical histological structure of an adenolymphoma yet was removed from the larynx.

The patient, a female aged eighty, had first noticed symptoms approximately four months before presenting for treatment. On examination a solid non-fungating growth 2.5 cms. in diameter was seen in the region of the left false vocal cord.

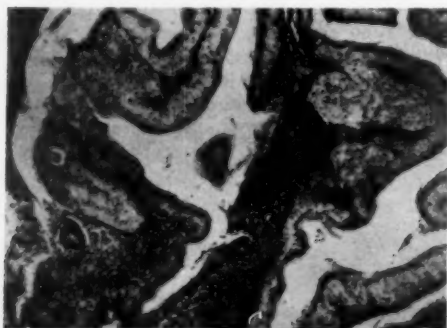


FIG. VI. Photomicrograph of a adenolymphoma removed from the false vocal cord. The characteristic epithelium and lymphoid stroma can be seen (x180.)

A biopsy specimen was taken and Fig. VI shows a photomicrograph of the tumour. It can be seen that the epithelium is of the same type as that described in the other twelve adenolymphomata and it has the same cystic and papillary pattern. The lymphoid elements, although a prominent feature, are not as abundant in this biopsy specimen as in other tumours and the epithelium is certainly not confined in a node of lymphatic tissue.

Approximately two thirds of the neoplasm was removed and when the patient was examined four months later the lesion appeared to be smaller. As this check on the patient took place only a few weeks before writing this report, the ultimate outcome is not yet known.

This laryngeal tumour is undoubtedly a typical adenolymphoma and appears to be arising from mucous glands. Although the "mixed salivary" type of growth occurs in many parts of the body adenolymphomata seem to have been previously described only in the neck.

It is interesting to note in this area the same relationship of lymphoid tissue and epithelium as is seen in the cervical region. The fundamental nature of this association is unknown but in the laryngeal growth it is certainly not due to the segregation of epithelium in a lymph node. These lymphoid aggregations are obviously controlled by the epithelium. Bloom (1945) has shown that, in certain circumstances, epithelium may even differentiate into lymphoid tissue.

The development of an adenolymphoma in the larynx, whilst not disposing of them all, does refute several of the theories advanced to explain the origin of these neoplasms and further discussion of such theories would not be profitable.

Actually, as indicated above, the problem of the genesis of the adenolymphoma is not of a sharply defined group but of a number of different types of growth one merging into the other. The various forms of parotid tumour constitute a range of tissue activity in which, unfortunately, certain examples have been given, and are still given special prominence. When it is appreciated that each is only part of a general group, it becomes apparent that many of the difficulties raised are artificial and in view of the plasticity of tissues, as exemplified by the laryngeal tumour described, are meaningless.

#### TREATMENT.

In most papers on adenolymphomata it is stressed that the tumours are easily enucleated but although undoubtedly true this is merely of academic interest as all neoplasms of the parotid should be excised. Adenolymphomata rarely recur even if enucleated but it must also be remembered that their preoperative diagnosis is very difficult unless a drill biopsy is obtained.

As under 100 cases of this tumour have been described it is difficult to assess the value of radiotherapy in their treatment.

Lederman (1943), who used the method, is of this opinion although it is clear that his cases were not typical.

Robinson and Harless (1943) state that radiation is of no value and as the results of surgery are extremely satisfactory it would seem to be unnecessary. Recurrence in the reported cases is rare and in those in which it was seen squamoid areas seem to have been present. In the present series of selected typical examples there is no known case of recurrence.

In conclusion it can be stated that the adenolymphoma, which is not as uncommon as the number of reported cases would suggest, holds most interest as a member of a group of salivary tumours rather than as an isolated phenomenon.

#### SUMMARY.

1. Twelve adenolymphomata are described and also what appears to be the first reported case of this tumour in the false vocal cord.
2. Some theories of origin are discussed and it is stressed that the problem of their genesis belongs to a group of neoplasms.
3. The tumour from the larynx serves to pin point the issue as it tends to focus one's mind in considering the plasticity of tissues.
4. Excision is eminently satisfactory as recurrence is rare and radiotherapy seems to play no part in the treatment.

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## RECENT CANCER RESEARCH AND ITS RELATION TO OPHTHALMIC AND OTHER CLINICAL PROBLEMS.\*

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Perth.

THE orthodox methods of treating new growths, namely by excision or by radiation or by both combined, leave much to be desired. We must realize that their success depends entirely on the stage at which the disease is first diagnosed. Newer chemotherapeutic and hormonal treatments show a trend in the right direction but have not so far achieved more than temporary alleviation. To understand the present situation we must deal for a moment with the history of cancer research in order to understand our own ideas and aims in treatment.

Our conceptions on cancer aetiology are founded on the work of a succession of famous German pathologists of the second half of the 19th century (before the era when the electron microscope, the chemical activators and the viruses were known) and especially on that of Ribbert. These celebrated pathologists separated cancer, by careful histological studies, from other more or less similar diseases: for example, it was once believed that coccidiosis of the liver in rabbits was a malignant growth.

Ribbert concluded after many years of study of metastases that the spread of cancer throughout the body depended on the extension of the growing tumour into lymphatics or blood vessels and the carriage of such cells to remote parts of the body, there to continue growth to form a secondary tumour. The process of growth therefore is unlike any infection: in Ribbert's famous saying a cancer grows not by infecting or rendering normal cells malignant, but *aus Sich heraus*, that is to say from its own resources. Hence if the primary growth is recognized early and completely removed before the malignant cells have passed beyond the site of origin, removal by operation is a simple easy cure. Once this stage is passed success depends on the relatively hit or miss irradiation of the secondary

field and cannot be guaranteed, since we can never know where each infiltrating cell has settled. We have always hoped to find a radiation specifically lethal to malignant cells and we have been encouraged by occasionally observing miraculous disappearances of malignant tumours following radiation therapy.

The introduction of radium needles and later of radon seeds has enabled us to apply this treatment with remarkable accuracy of positioning but its success still depends on the lucky diagnosing of the growth before metastases have occurred. For example, the use of radon seeds in the eye, introduced directly into neuroblastomata or other intra-ocular tumours through a scleral trap-door or stitched into position on the outside of the globe, has revolutionized an almost completely hopeless outlook, though the local success of the operation must not deceive us into thinking the patient is necessarily cured.

Most of our accurate knowledge of cancer, apart from the minute histology of the human tumours, comes from animal experiment. The pioneers of experimental research in cancer—C. O. Jensen of Copenhagen, E. F. Bashford, the first Director of the Imperial Cancer Research Fund, Paul Ehrlich, Borrel in Paris and Leo Loeb in the United States—quickly established that cancers of mice and men arise, grow and behave similarly, and are in fact one and the same disease. They arise more frequently in the old than in the young, they grow from their own resources—*aus Sich heraus*—they form metastases in the same way as human cancer does. They are readily curable by early excision of the primary tumour; some, as was found by Bashford, are radiation sensitive, the majority resistant.

\*Read at the Annual General Meeting, Sydney, June, 1951.

Some of the experiments which have had the most persistent effect on world medical opinion and outlook, were carried out in the laboratories of the Imperial Cancer Research Fund by Bashford and his assistants. These must be described briefly since the conclusions drawn from them have had such a deep and darkening influence on cancer research the world over.

It was found by the pioneer investigators, even as early as 1820, that cancers of men cannot be transferred to dogs; in the early years of this century attempts were made by several surgeons to graft a tumour of man under the skin of another man: in each instance the surgeon himself played the part of guinea-pig. In one such example the graft of the tumour grew and had to be excised. Jensen, Bashford and Ehrlich established the fact that mouse cancers do not grow in rats nor rat cancers in mice. In fact the species barrier is absolute in cell transplantation. The experiments referred to above, which appeared to give some meaning to these findings, were designed to find out what happened to the cancer cells when a small piece of a carcinoma of a mouse was implanted under the skin of another mouse. It was found that all the connective and vascular stroma of the fragment—that is to say, the normal cells of the implant which constitute the framework and food supply to the tumour—die rapidly; next, the central mass of cancer cells dies, the outer shell of the implant alone remaining alive. Angioblasts and delicate connective tissue cells provided by the new host invade the graft and the surviving cancer cells grow and multiply and form the daughter tumour. These experiments were repeated again and again and always with the same result in carcinoma, where the malignant cells can be readily distinguished from the connective tissues into which they are grafted. When a sarcoma is examined it is not possible to determine with certainty whether the daughter tumour is derived from the implanted cells. It appeared to Bashford and Murray to fit closely into Ribbert's conception of cancer and agreed so perfectly with the facts that no micro-organism could be revealed in tumour cells by the then best

available microscopes—that the passage of tumours from animal to animal could be achieved only by the insertion of living cancer cells into animals of the same species and variety as that in which the primary tumour arose, and that cell free extracts and dried tumour tissue failed to start a new tumour, that there was no escape from the conclusion that the intracellular cause of cancer is a "distortion or alteration of a mysterious kind" of the normal cell when it becomes malignant. Bashford, Ehrlich and the early students of cancer realized very clearly and stated again and again that the aetiology of new growths is divisible into two parts, namely the remote causes and an intracellular or "continuing" cause.

Of remote causes few were then known, compared with the present state of our knowledge; there were soot, proved to be carcinogenic by the surgeon Percival Pott of St. Bartholemew's Hospital in 1775; coal tar, proved by many clinicians who practised in areas where tar and pitch were used to bind coal dust in the manufacture of briquettes; bilharzial infestation of the bladder and rectum; prolonged application of heat to any area of the skin, as for example the Kangri cancer of the skin of the abdomen in Indians, and X-rays and radium emanations.

From none of these cancers or from cancers induced in rats and mice by similar means could the remote or initiating cause be recovered again. It would not be expected for example, that X-rays could be obtained again from an epithelioma arising in a dermatitis occurring in a radiologist several years after he has ceased working with X-rays. Nor can soot be recovered from the metastases of a "soot epithelioma." It was well understood by Bashford, Ehrlich and all the pioneers of cancer research that these causes—chemical or physical or parasitic—are merely remote or precipitating or initiating causes, which, having started a cancer, are no longer necessary for its continuation, its cell multiplication and spread, either in the original victim or in the case of animals, in subsequent hosts.

The aspect of aetiology which interested these workers, the pioneers of research in cancer, is that which was always regarded as the intracellular or continuing cause. But no such intracellular cause could be found at that time and all the superficial knowledge then available appeared to support the conclusion that the continuing cause depended upon some mysterious change in the structure of the malignant cell itself. This positive conclusion, based on purely negative evidence, was propagated with a vigour more often found in religious or political subjects than in scientific affairs. It is recorded that Ehrlich, one of the greatest of medical investigators, remarked to a young doctor who was anxious to do cancer research, that it was a foolish undertaking, that we should make little advance in knowledge until we knew completely the chemical and physical activities of normal cells—in his words “the secret of life itself.” He said that he himself had wasted many years in cancer research and finally advised the aspiring student to give up his plans.

When we contemplate the enormous world wide organisation which has grown up around cancer research, the vast buildings, the hospitals, laboratories, centres of cancer education and propaganda both in the Commonwealth and in the United States, we cannot but be surprised and chagrined to find in how many researches the primary object, namely the understanding of the continuing cause, has been lost sight of. This has come about largely from too blind an acceptance of the “cell distortion” theory (which postulates that the unit of the disease is the entire cell itself). We are reminded of Wilfred Trotter's essay entitled “Has the Intellect a Function?”, in which he points out with great subtlety the fact that the intellect is mainly used to rationalize belief. He remarks that “great discoveries will therefore continue to be unexpected—and that research deliberately directed against short range targets is apt to be held up contrary to all reasonable expectations,” and recommends the contemplation of the “bitter war of attrition that has marked the enormous and world wide attack on cancer.”

But this is too gloomy a view of the situation, as we shall see if we consider certain factual advances which have been made within the last 30-40 years and which, when taken together, begin to fall into a simple and logical pattern, largely made possible by coincident advances in other fields, in the light of which they become intelligible. We will consider these related advances first. They centre round increased knowledge of certain bacteria, including knowledge of chemical activators, increased understanding of viruses, especially of non-pathogenic phases of pathogens, increased facilities for examination due to the electron microscope, and increased understanding of the thermal properties of viruses and of their facility in mutation, largely due to the study of phage and varicella. Research proceeding along a rigid furrow without contact with parallel lines of work is no longer very fruitful in any branch of knowledge and cancer research is no exception.

#### ACTIVATORS.

By activation is meant conversion of the latent non-pathogenic phase of an infecting organism into the pathogen by chemical or physical agents. Tetanus and tubercle bacilli and the virus of herpes febrilis can be used as examples. It is now well known that washed live tetanus spores can be injected into guinea-pigs and that no clinical tetanus will develop (Bullock and Cramer, 1919). If even after months, such guinea-pigs receive, at the site of injection, an injection of a solution of calcium chloride, acute tetanus will develop in every case within a few days. The calcium salt is as necessary to produce the disease as is the bacillus, since this is not apparently able to multiply and to manufacture its toxin without the chemical activator. A similar relationship has been demonstrated between the tubercle bacillus in the lung and silica sol—well exemplified in the pathology of miners' phthisis. Among viruses similar reactions are found. The virus of herpes febrilis is known to enter the cells of the muco-cutaneous junction of the lip very early in life but to remain in the latent phase, multiplying *pari passu* with the cell,

until activated by heat (either a rise in the host's own temperature or exposure to external heat) when its rate of multiplication increases sufficiently to disrupt the cells and produce the clinical manifestations of herpes.

The electron microscope has rendered visible many objects whose presence had previously been inferred from experiment. It has shown among other things that viruses have a distinct structure and shape, which varies from group to group, and in this way strengthens the case for their extrinsic origin and separate existence. This controversy (intrinsic or extrinsic origin) need not concern us, though if we require a lead on the matter we have only to consider the natural history of diseases such as measles and influenza to feel that an intrinsic origin has never been proved.

#### PROPERTIES OF VIRUSES.

The difficulty of keeping viruses alive outside a living cell (host animal or egg membrane) has led to extensive studies of their thermal properties and it is now known that most of them are highly thermolabile but appear to go into a resting phase in extreme cold, so that they can be kept "alive" at temperatures (even as low as that of liquid air) which would immediately be lethal to the host animal, whether vertebrate or invertebrate. As well known but comparatively recently discovered examples, we can mention typhus and scrub typhus, which have to be kept at temperatures well below freezing. Some of the larger viruses, such as that of variola, can withstand relatively higher temperatures — otherwise vaccination with calf lymph at room temperature would not be successful. Indeed each virus appears to have its optimum temperature for extra-cellular survival, but these temperatures are on the whole much lower than those of survival of multicellular organisms and often lower than those of bacteria.

Other properties of viruses which we must consider are their capacity for existing intracellularly in a latent phase for long periods, and their capacity for sudden mutation. Each virus specializes in life in a definite type of cell. The herpes febrilis virus prefers the

epithelium of a muco-cutaneous junction, where it may remain latent for years. The varicella virus prefers epithelial cells, its herpes zoster mutant is highly selective for posterior root ganglia. The poliomyelitis virus, which probably normally lives a harmless existence in cells of the alimentary tract, may suddenly mutate to the serious central nervous system variant with highly selective preferences. Even in the eye we are struck by the fact that chronically recurring herpes of the conjunctiva practically never shifts either to the cornea or to the skin of the lid. *Per contra*, dendritic ulcers of the cornea do not invade the conjunctiva.

#### LIFE CYCLES OF VIRUSES.

It is now known that many viruses (e.g., psittacosis) go through a series of changes of size and shape and infectivity which constitutes a definite life cycle comparable to that now known for many bacteria (e.g., tubercle) and parasites (e.g., malaria). The simplest form of this cycle is the alternation of a latent and an active phase (e.g., phage). The reason for the change from one phase to another is seldom understood, except in the cases where an extrinsic activator has been discovered (e.g., herpes).

These observations may appear to have little to do with the problem of the continuing cause of cancer until we realize that in all the innumerable tumours studied in laboratories the world over, and induced by innumerable chemical and physical means, no single initiating cause has ever been recovered from the *n*th transplant of a tumour and been found to be capable of initiating a similar tumour afresh, apart from one outstanding exception. This exception is a virus.

#### VIRUS TUMOURS OF BIRDS.

The discovery of the virus of chicken sarcoma by Peyton Rous in 1911 provided this, the first exception to the rule that tumours could be propagated only by grafts of living cells. Since 1911, from over two hundred different tumours of birds (spindle-celled sarcoma, myosarcoma and endothelioma among others) viruses have been recovered which will start a new tumour when

injected into a new bird of the same variety. Freezing, drying and filtration methods of separation of the virus from the living cells are equally successful and much is now known of the properties of such tumour viruses, their cellular preferences, their thermal preferences, their alternating phases of high and low activity, their electron microscope appearances and their immunological reactions. There is strong evidence that during rapid growth of the tumours the virus particles acquire a nucleo-protein envelope (Gye and Purdy, 1931), which enables them to pass into the nucleus and by irritating this, stimulate the cell to divide (without disorganising it entirely as does merely cytoplasmic multiplication in most other virus diseases). In Theobald Smith's famous phrase they "are examples of perfect parasitism" which accounts for the fact that the disease is not self-limiting.

It would appear therefore that the viruses of the chicken tumours afford the first instance of the isolation of the continuing cause of the cell multiplication in cancer.

It was at first thought that these tumours formed a group apart but since 1911 the number of tumours of which viruses form the continuing cause has increased and has culminated in the discovery of a widespread group in mammals which must be considered in detail.

#### VIRUS TUMOURS OF MAMMALS.

The first mammalian virus tumour to be discovered (in the early 1930's) was the Shope virus papilloma of wild cottontail rabbits. In this case the tumour is clinically an innocent one as it does not metastasize, but under experimental conditions in laboratory rabbits a malignant change occurs and the papilloma cells become epitheliomatous. The Shope virus resembles the Rous virus in its filtrability, its transmissibility by frozen and dried material, its visibility with the electron microscope and its invariable recoverability from the papillomata of cottontail rabbits.

The second virus mammalian tumour known is the Bittner mammary adeno-carcinoma of mice. The story of its discovery is

complicated by the apparent genetic aspect of the earlier experiments. The use of genetics in the study of cancer dates from the work of Maude Slye and Leo Loeb in the United States on the production of high and low cancer strains in mice rendered practically homozygous by continuous brother-sister mating. Some of the strains so produced showed a 97 per cent. death rate in the females from cancer of the breast, while other strains showed no cancer at all. It was naturally concluded that this proved the genetic presence of a tendency to malignant change in breast tissue; but it was later proved by Loeb that another factor was required. He found that spayed females did not develop cancer and that males given oestrin for long periods developed it even more readily than the intact females. To an apparent hereditary tendency it was therefore necessary to add a chemical activator—oestrin.

The whole genetic basis of the conception was, however, destroyed when it was discovered in 1936 by Bittner at Bar Harbour, Maine, that the young of the high cancer lines, if suckled from birth on mothers of strains free from cancer, did not themselves develop cancer and that from their offspring could be bred out a cancer-free strain genetically identical with their parents. Thus was discovered the "milk factor" or Bittner virus of which the life history is now well known.

This virus exists in two forms, latent and active. The latent form (visible with the electron microscope as a minute sphere) can be found throughout the bodies of both the male and female mice of the affected strains throughout their lives, and also in the milk of the females. The latent form is non-pathogenic. The active form, visible as a minute sphere surrounded by a thin envelope, possibly nucleo-protein in nature, can be found only in the cells of the tumours (Porter and Thompson, 1948). Conversion of the latent to the active form occurs under the slow activating influence of oestrin, either the females' own oestrin or administered oestrin in the males. Tumours then develop. From these tumours it has recently been shown that new tumours can be started

afresh with frozen or dried material (Mann, 1949; Mann and Dunn, 1949), provided this is injected into mammary tissue, since after freezing to  $-79^{\circ}\text{C}$ . the material becomes tissue specific. Filtration has not yet been achieved since centrifuging experiments with long frozen material in which the cytoplasm has been destroyed show the infecting agent to be within the nuclear membrane (Gye *et alii*, 1949) and this presents technical difficulties. Material frozen at or below  $-79^{\circ}\text{C}$ . for periods up to a year or more increases in infective activity during the first three weeks, presumably owing to slow disintegration and denaturing of the tumour cells and freeing of the virus. The virus is extremely thermolabile and cannot be handled above  $-10^{\circ}\text{C}$ . when infectivity rapidly falls. Drying must be done at approximately  $-25^{\circ}\text{C}$ . *in vacuo*. The virus can be recovered from the *n*th generation living transplant in male mice in the complete absence of oestrin, and therefore represents the "continuing cause" of mammary adenocarcinoma in mice, oestrin being the initiating cause or activator.

Thus a new large group of tumours has been brought into line with the Rous chicken tumours. In addition, Lucké has discovered a virus tumour of frogs, and more interesting still, Gye *et alii* at the Imperial Cancer Research Fund, have shown that sarcomas of the connective tissue of mice (even in strains free of the Bittner virus), either arising apparently spontaneously or induced by means of coal tar derivatives, can be propagated by long frozen and dried material. Thus we find a virus as the continuing cause of many hundreds of avian, amphibian and mammalian tumours. Much more work is needed, naturally, to demonstrate its invariable presence in all tumours, to explain its various modes of entry to the body and its apparent endless capacity for mutation. We have also to seek the explanation of the action of the hundreds of initiating causes or activators, though these, being all irritant rather than destructive, probably merely allow the latent form to acquire a nucleoprotein envelope and so enable it to stimulate the nucleus to divide.

#### PRACTICAL APPLICATION.

As Wilfred Trotter remarks in his essay "De Minimis" the law does not concern itself with small matters: *de minimis non curat lex*. It is, however, characteristic of science that the smallest apparent exception must be considered: *de minimis curat scientia*. We are faced in the clinical study of cancer with certain apparent rules and with certain minute exceptions to them. If these minute exceptions can be explained by new experimental facts they form ancillary evidence for the truth of these and help to unify the clinical and pathological picture. One of these clinical rules about cancer is that any organ or tissue containing cells normally capable of division may undergo malignant change. If the cell is fully differentiated (as, for example, the "noble cells" of the brain) it cannot form a tumour. This seems to be in accord with the cellular or cell distortion theory of cancer until we notice some minute exceptions. The lens of the eye shows mitotic figures in its subcapsular epithelium even up to the age of 80 years, the *substantia propria* of the cornea contains cells capable of migration and of repair activities throughout life, the internal coats of the large blood vessels contain active fibroblasts, yet in none of these places does a malignant tumour ever arise. The lens never gives rise to an epithelioma, corneal neoplasms always arise from the limbus and invade the *substantia propria* secondarily. Why these minute exceptions to the general rule? Science must take note of them.

If we consider these exceptions we shall see that the lens and the cornea have no blood supply and never have had throughout development, the intima of the great blood vessels is avascular and, pursuing the idea of vascularity, we see that the more vascular an organ up to a point the more prone it is to malignant change. Hence sarcoma of the sclera is a great rarity and, when it occurs, often arises at the insertion of a muscle where the blood supply is slightly greater. One might object that in these exceptions the tissues owe their immunity to the difficulty of an initiating cause reaching them, but it is obvious that this will not hold for the eye. Many chemical activators are easily

diffusible and physical ones, such as ultra-violet light would find no difficulty in reaching the lens. Indeed it is possible to produce changes in the lens with X-rays but the change is never a malignant one.

In the case of the lens the position has been clarified experimentally. Chemical activators injected into the lens *in situ* do not give rise to epitheliomata, but if a young lens is implanted under the skin of a mouse known to carry a cancer virus in its blood stream and is treated with a chemical activator (after rupture of its capsule) it is possible to produce an epithelioma of the sub-capsular epithelium. This demonstrates the three essentials for a tumour: a cell capable of division, a blood supply containing virus in its latent form, and an activator. In the case of the lens the production of a tumour is not easy. Many trials must be made. This is probably due to the unlikelihood of a virus mutant capable of living in lens epithelium reaching the implanted lens in every case and shows that blood supply and activator alone are not sufficient.

Let us now consider from these premises what parts of the eye we might estimate as likely to be immune or prone to cancer. The lens as we have seen, owes its immunity to complete lack of blood supply, the centre of the cornea likewise, limbal growths arising at the point of greatest vascularity. The sclera has a poor blood supply and is proportionately almost immune to malignancy, which when it does occur tends to do so early in life. All these facts strongly support the view that cancer viruses reach the affected organs via the blood stream in early youth.

We should now consider the converse proposition. What parts of the eye would be expected to be immune to cancer on account of their complete state of differentiation? The *pars optica* of the retina has differentiated by the end of the 6th month of life and none of its cells are capable of dividing again, except occasionally a few astrocytes in the glial supporting tissue. What is more, practically all the differentiation occurs before a blood supply is developed at all. Indeed the spread of the blood vessels is from the disc toward the periphery as is

the differentiation also, this latter preceding the vascularization by many weeks throughout development. The only situation where development lags behind is the fovea and this is devoid of blood vessels throughout life. Malignant tumours of the retina would therefore be expected to be rare (as they are) on account of the fact that differentiation is practically complete before a blood supply appears. They would also be expected to occur early in life or even before birth (as they do) since only then could the cells involved divide. The retinal mutant of the virus may be conveyed through the placenta. It may also be that in the affected cases differentiation had lagged behind vascularization slightly. The placental passage of virus would account for the familial incidence; the only genetic possibility is that of an inherited lag in differentiation which would allow the blood supply to reach undifferentiated cells. With the story of the Bittner milk virus in our minds we must be chary of postulating genetic influence in familial neoplasms.

True astrocytomata of the retina are rare and may arise at any age. Tumours of the hexagonal epithelium are unknown and this differentiates very early. Malignant growths, carcinomata, of the ciliary body occur but are rare. One, the diktyoma, arises early in infancy or before birth. The true ciliary epithelioma, however, occurs in adults and this is not surprising when we remember that the ciliary epithelium can hypertrophy in case of injury or inflammation and the cells are thus known to be capable of division throughout life.

The common malignant melanoma of the choroid presents no difficulties as it arises often from the ectodermal Schwann cells lying in the extremely vascular choroid. Cases of apparent inheritance of malignant melanoma are known. One family, reported by Davenport, shows the passage for three generations through affected females, the offspring of unaffected individuals being normal. This will fit equally well into a genetic theory or into the picture of a virus mutant conveyed from mother to child. Tumours of the iris and choroid proper are also as simple of explanation as connective tissue tumours elsewhere.

We thus see that by linking the study of cancer and ophthalmology and concentrating particularly on the minute apparent exceptions further support is gained for the idea of a quadruple trigger mechanism in cancer: a cell capable of dividing, a blood supply, a virus and an activator, intrinsic or extrinsic. Naturally there are many queries unanswered still but they merely now point the way for further experiment.

These examples taken from ophthalmology might well be extended to include other specialties in which the same connection between blood supply and state of differentiation will be found to hold good. Intracranial neoplasms afford a good example of the immunity of fully differentiated cells, as do tumours of the teeth and jaws. Tumours of dental enamel and dentine are pathological curiosities since here both early differentiation and poor blood supply are present.

In some cases an organ, such as the heart, may owe its relative immunity simply to the rarity of a virus mutant capable of living in that type of cell. Indeed such a mutant might conceivably have the superficial appearance of a "lethal gene" since it would tend to the disappearance of the stock carrying it. We know of examples already in which so-called lethal genes have turned out to be viruses (e.g., the "killer gene" of a paramoecium).

But to continue in a still more speculative vein. How does this new knowledge affect us as clinicians? In the first place it brings order into chaos and removes the mysterious, incomprehensible, unsatisfying standpoint that tumours are "causeless" or due to "cell distortion" or "form a problem in energetics" as has been stated by one author, all of which phrases when examined become purely negative and hopeless. It allows us a clearer mental picture of how to tackle the disease. In the first place one can seek to tackle either the activator or the virus, since the virus in its latent form hurts no one. It is, however, at once obvious that we cannot remove the activators since they are legion. We can, however, curb them as we do when we protect our X-ray workers, when we advise tannic acid against sunburn in susceptible individuals, when we seek to

remove from industry the more obvious of the carcinogens or at least to warn and protect the employee.

But how can we tackle the virus? If it could be removed the activators would be harmless. We can in theory attack it at three points namely, before it enters the body, while it is still latent, and after it has changed into its active form and become manifest. To seek to prevent it entering the body appears at present hopeless, since cancer viruses are so wide spread as to appear almost universal. It is true that some families appear to carry one mutant rather than another, for example, the families with apparent dominance of malignant melanoma of the choroid or neuroblastoma retinae or mammary cancer. But studies of in-bred mice have shown that several mutants may be present together and that some may remain latent for generations. Again, we do not know how the virus passes to the offspring. We know that the milk is one avenue of entry and there are many others. Indeed the latent form can probably be carried as a passenger by the sperm. Therefore antenatal or natal attack is not advisable. Indeed in spite of evidence in in-bred mice, cross suckling or artificial feeding of human babies does not entirely remove the risk of breast cancer (Horne, H. W., 1950). The advantages of breast feeding probably outweigh entirely the elimination of one only of the possible portals of entry.

There remains, therefore, the possibility of killing the virus in its latent form when it is disseminated throughout the body and being carried by the blood stream. No work has so far been done on this but there are possibilities for chemotherapy here which are well worth exploring.

Finally, if cancer is a virus disease it should be possible to find a substance selective for the group of cancer viruses. Until the discovery of chloromycetin just before the end of the war, no substance was known which would attack viruses, though many had been prepared against bacteria. With the discovery of the antiviral antibiotics, all, so far, it is true, active against acute virus infections only, we enter a new era of hope. The problem is, of course, to find a substance, non-toxic to the host, toxic to the virus and capable of penetrating the nuclear

membrane and attacking the intranuclear active form of the virus. From the evidence we have at present at our disposal the *Streptothrix* group of organisms is most likely to provide the desired antibiotic. Indeed one such, having a definite action on certain tumours of mice is already known (de Angelis, 1949). Work on this and allied substances is already in progress in Europe and in Australia and is well worth extended effort.

From the clinicians' point of view the advantage of such a substance over surgery and irradiation would be its selective power of attacking metastases wherever they occurred. It is true that chemical treatments such as oestrin for prostatic cancer and radio-active iodine for malignant thyroid also attack metastases but the underlying principles are different. In the first case use is made of a physiological principle to hold the cell in check but not to kill the virus. In the other the action is destructive and selective and more like that aimed at in

an antibiotic. In all these cases, however, the action is palliative rather than curative. There is in the present state of our knowledge no inherent impossibility in the idea of finding a true cure for cancer provided we reorient our ideas on aetiology in accord with experimental evidence and coincident advances in knowledge in other branches of pathology.

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## BONE MARROW AS A COMPONENT OF BONE SARCOMA.\*

By E. S. J. KING.

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*The hard and soft, seem all affin'd and kin:*

*Troilus and Cressida. Act. 1. Sc. III.1.25.*

A GREAT deal has been written on the subject of bone tumours, even though they are not common; indeed if the sheets of manuscript were placed end to end they would pave a road from Melbourne to Sydney. At the same time, in the last few years little has been added to our knowledge of them. It is not proposed to-day, therefore, to merely restate what has been said already but to discuss some points in the pathology of these tumours which either have not been considered or have been given insufficient attention.

The subject of bone tumours is far too large to be encompassed, even in a sketchy manner, in the time at our disposal so that it is proposed to deal only with some features of osteogenic sarcoma.

Prior to 1927, when Kolodny reviewed the specimens of the Codman Registry of Bone Sarcoma of the American College of Surgeons, ideas regarding bone sarcoma were incomplete and poorly defined. There were many "classifications" depending on special features—macroscopic structure, radiological appearances, histological form, vascularity and the like. Histologically a great many different kinds of tumours were described according to the tissues found in them.

Kolodny's contribution was the emphasis of what had been becoming appreciated generally, that the various connective tissues were closely inter-related and were often transformed one into another. The idea that a fibroblast could form only fibrous tissue was waning, and, similarly, the view that bone could be formed only by a specific cell—the osteoblast—was also fading.

By definition, the bone-forming cell is an osteoblast, but that an "osteoblast" might in certain circumstances form fibrous tissue, cartilage and mucoid tissue amongst others

is an indication of and emphasizes the limitation of definitions which take cognisance only of a limited range of activity.

Since the various components of the tumour are closely related it is not so necessary, as was once thought, that they should all be specifically mentioned, for example in a diagnosis such as chondro-myxo-osteosarcoma. This problem of terminology was overcome by the use of the term osteogenic sarcoma which had been suggested by James Ewing. All the connective tissues are osteogenic, just as they are fibrogenic or chondrogenic, so the term had a clear meaning and its brevity conveyed great advantages.

Bone is the most impressive connective tissue because of its hardness, its durability and relative opaqueness to X-rays. Thus it is natural that the bony component of the tissues should be emphasized (etymologically) to the exclusion of the others. This is an important point because often there is little or no bone in the tumours though clearly, in the macroscopical sense, they arise from the bone and histologically the relation of the various tissues has been shown.

Classifications based on the microscopical structure of tumours may be considered principally from either the histological or the histogenetic viewpoint. An histological grouping has a sound observational basis whereas the histogenic classification depends on an assumption that the appearances of cells must necessarily be a direct indication of their origin.

Some writers (for example, Willis, 1948) appear to dislike the term osteogenic sarcoma and prefer the simpler term osteo-

\*Read at the Annual General Meeting, Sydney, June, 1951.

sarcoma. The use of such a term is based on the acceptance of an histogenetic classification. I would emphasize here emphatically that any histogenetic classification presupposes knowledge of the origin of tumours that we cannot possess. If the method of classification be an histological one then the term osteosarcoma is open to many objections because the tumours, as mentioned earlier, often contain tissues other than bone.

It is important to reiterate that when we see a well-developed tumour (and we usually observe growths in the well-developed stage) it is not possible by any means at our disposal at present to determine the cell or cells of origin. Too many cell divisions, with the possibilities of differentiation in various directions, have occurred for an opinion as to the form of the originating cells to be proposed with any certainty. We know the organ of origin usually but it is assumed that the cells of the tumour came from cells of a kind similar to them. This is probably true but nevertheless is speculation. What is beyond question, however, is the structure of the cells of the growth and when the tumour is named after them—as indeed it usually is—we are on safe ground.

At the same time most of the bone tumours show some histological complication of structure and it is most undesirable that we should return to the old complex (and unnecessarily multiloquous) terminology. Provided that the capacity of any of the connective tissue cells to produce different tissues is completely appreciated the term osteosarcoma could be employed (in a peripatetic histological sense) but personally I doubt whether we have yet advanced to a stage where some at least will not find it misleading. For this reason the term osteogenic sarcoma is still employed here.

One special feature of bone tumours which has been remarked by very few observers is the presence of bone marrow tissue as a component of them. That bone marrow may be found in many parts of the body is well recognized. It is sometimes said that it develops from reticular cells and certainly it is found specially in such places as the spleen, liver and lymph nodes, in which cells which we regard as reticular are numerous. At the same time there is

good evidence that reticular tissue is co-extensive with other connective tissues and indeed the reticular cell is closely related to the endothelial cell which in turn is a close relative of the fibroblast.

When heterotopic bone is formed it usually shows bone marrow tissue between the trabeculae after it has reached a certain degree of organization. Such bone is found in most parts of the body, common examples being the wall of the aorta, the bronchus, in the skin amongst other unusual sites. Although a century ago it was suggested that it arose by growth of cells metastatic from the bone marrow of the long bones this is not now even considered; it is recognized that bone marrow may develop in any part of the body and, in the less usual sites, specially in relation to heterotopic bone.

Tumours composed of bone marrow tissue occur as the various forms of myeloma and related growths. These are found usually in the skeleton but may be found elsewhere. These are related to the bone tumours proper in two main ways. (i) Tumours composed of reticular tissue—the reticulosarcomata of the bones—may be regarded on one hand as an anaplastic or “undifferentiated” form in the myeloma series or on the other as a non-osseous type of bone sarcoma. (ii) Recognizable bone marrow tissue may be encountered as an integral part of a bone sarcoma. It is the second group which is to be discussed here.

Occasional examples of tumours containing bone marrow elements have been described (Rodme and Delaney, 1932) but these are only few and the importance of the association does not appear to have been appreciated. The writer has encountered several examples both in tumours showing good differentiation in the direction of the connective tissues and those which are relatively poorly “differentiated.”

A case of osteolytic sarcoma, in which a large element was demonstrably of haemopoietic tissue, illustrates the phenomenon very clearly and is worthy of description.

#### CASE HISTORY.

R.H., male, aged 27 years, began to have pain in the left leg immediately below the knee. At first intermittent, this soon became constant and was

worse at night. When first examined no abnormality was detected but, after three weeks during which the pain remained unabated, he was sent for specialist opinion.

At this time (seven weeks after the onset of symptoms) there was some slight swelling of the upper part of the tibia on the outer aspect. There was some increased heat over this area. X-ray examination showed an area of rarefaction in the outer portion of the upper part of the tibia immediately below the condyle. There was a soft tissue shadow contiguous with the zone mentioned between the tibia and fibula. In this tissue there were irregular spiculated fragments of radio-opaque material. The fibula was not obviously involved (Fig. I).

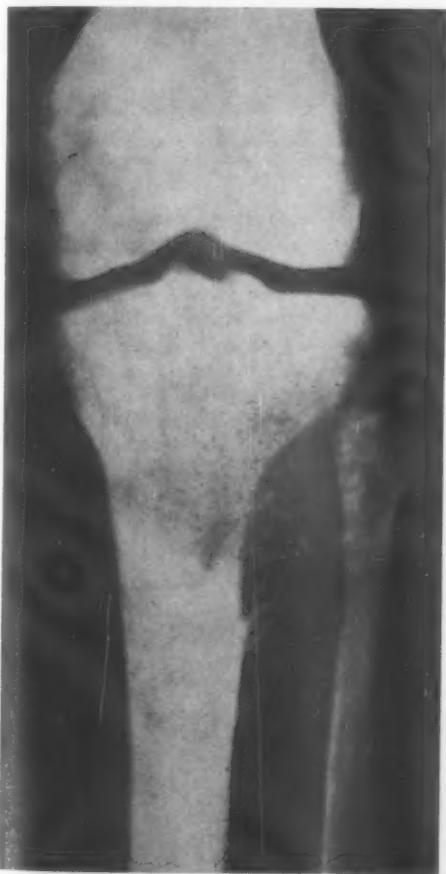


FIG. I. X-ray showing the tumour of the upper part of the tibia. There is rarefaction of the bone and a soft tissue mass, between the tibia and fibula, showing spicules of calcified material.

Investigation of the peripheral blood showed a mild anaemia (ranging from 3,500,000 to 2,500,000 corpuscles per c.mm. over a period of three months), a platelet count within normal range and a white cell count of 6,000 corpuscles per c.mm. There was a definite shift to the left of Arneht Index but no atypical white cells and no nucleated red cells were seen.

Amputation of the limb was performed but pulmonary metastases became apparent within six months of operation and the patient died just over twelve months after the onset of symptoms.

#### *Pathological examination.*

There was a mass attached to the outer aspect of the upper part of the tibia projecting into and displacing the muscles lying between the tibia and fibula. It was four inches long, two and a half inches wide and projected for two inches above the surface of the tibia at its most prominent part. When sectioned the bone was found to be replaced by a vascular, cellular and largely non-osseous tissue half way through the shaft and from the condyle for three inches down the shaft. Externally there was a fibrous capsule separating the growth from the muscles and internally the tumour tissue was sharply defined, macroscopically, from the neighbouring bone.

Histologically the tumour was extremely vascular and consisted mainly of spindle cells, some of these lining blood vessels but others being arranged irregularly. There was great irregularity in size and some variation in the shape of the cells; some were tumour giant cells (that is to say they contained a few large nuclei comparable in size with those of the surrounding tumour).

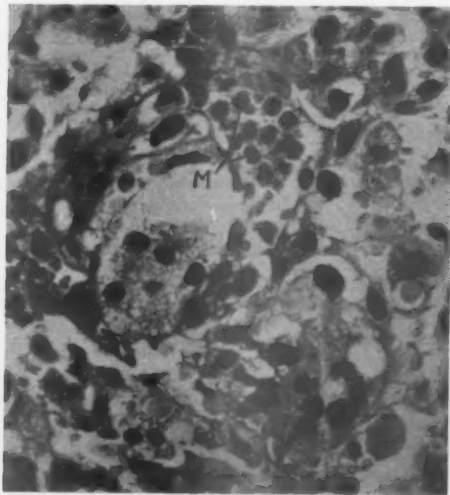


FIG. II. Photomicrograph of a section of the tumour showing a group of cells of the myeloid series amongst tumour cells. (x 320.)

Scattered through the tissue there were a few bone trabeculae and numerous foci of haemopoietic cells of both the red and white series (Figs. II and III). The haemopoietic foci were arranged independently of the bone tissue. Both bone and bone marrow were found in the part of the tumour which was topographically extrasosseous as well as in the part of the growth that was near the normal bone; that is, they appeared to be an integral part of the tumour. Some of the marrow cells were typical in form but there were some which were clearly different mainly in size, from any normally seen in the marrow (Fig. IV).

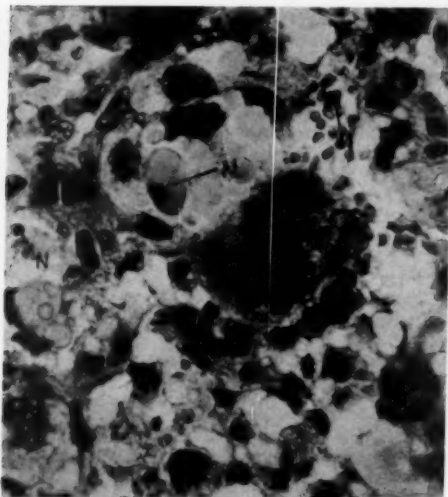


FIG. III. Photomicrograph of section of the tumour showing white cells and an atypical normoblast (N). (x 320.)

Examination of the bone and bone marrow of the skeleton of the limb, macroscopically and microscopically, did not show any deviation of the haemopoietic tissue from the normal pattern.

Metastatic tumours were observed radiologically in the lungs but no post mortem examination of these was made.

#### DISCUSSION.

When haemopoietic tissue is present in a tumour of this kind it is essential to determine whether such tissue be included accidentally in the growth during its invasion of the marrow, whether it arises as an incidental non-neoplastic metaplasia or whether it be an integral part of the growth.

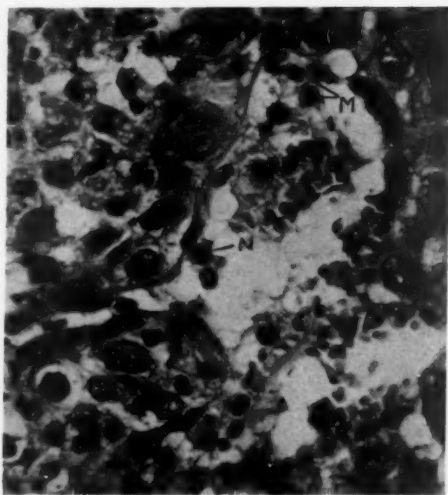


FIG. IV. Photomicrograph of a section showing normal and a grossly atypical example of nucleated haemoglobin-containing cells. (x 320.)

In the present case the presence of the foci of recognizable haemopoietic cells throughout the whole of the tumour even to the periphery and their intimate intermingling with the tumour cells throughout (Fig. V) make it most improbable that the foci (and in some areas scattered cells) are due to accidental inclusion. Inclusion therefore may be disregarded.

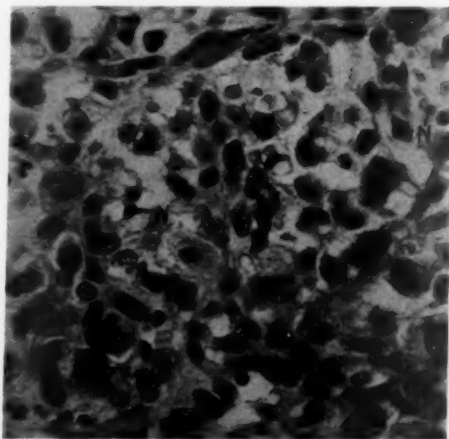


FIG. V. Photomicrograph showing cells of the white and red (N) series scattered amongst the tumour cells. (x 320.)

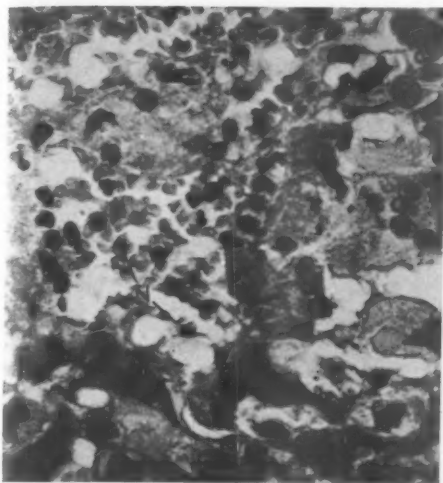


FIG. VI. Photomicrograph of a section showing bone marrow cells; nucleated haemoglobin-containing cells shown at (N). (x 320.)

Heterotopic development and hyperplasia of bone marrow in the stroma of the tumour, as a superadded phenomenon, is possible just as it may occur in other tissues; but unless there is evidence of widespread (that is generalized) haemopoiesis, the suggestion of a local development of bone marrow (apart from the tumour but co-extensive with it) is unjustified conjecture. More positive evidence is the nature of many of the cell forms observed. Though morphologically normal nucleated red cells and white cells (myelocytes and myeloblasts) were to be seen in large numbers (Fig. VII) there were several which differed so greatly in form (Fig. IV) from the normal as, in themselves, to make the idea of a simple hyperplastic stimulus unlikely and to indicate that the stimulus is within the range of neoplasia.

The blood examination carried out before and after removal of the tumour failed to show any significant change beyond the mild anaemia and the moderate shift to the left of the Arneht count. This, with the result of examination of the bone marrow of the bones of the limb, indicated that there was no great general haemopoietic activity. At the same time any atypical blood cells

formed in the tumour were not escaping into the blood stream in sufficient numbers to be observed in blood films.

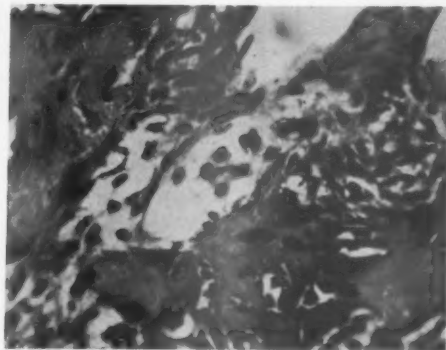


FIG. VII. Photomicrograph of a section from an osteoplastic osteogenic sarcoma with marrow cells (including some differentiated white and red cells) between the masses of osteoid tissue. (x 200.)

In this case the tumour was definitely osteolytic. Any tumour composed of non-calcified tissue, of course, will be bone-replacing in character—the degree depending on the proportion of non-bony tissue. It is not necessary, however, that marrow-containing bone tumours should all be osteolytic in type. In the present case almost all of the tissue is cellular with but little intercellular substance, and thus very little calcified tissue is present. In other cases, however, haemopoietic tissue has been observed within a bony or osteoid framework and thus the growth has been osteoplastic (Fig. VII). The case described here was chosen from a series because it showed the features most clearly.

Haemopoietic tissue may be found thus in bone tumours of all kinds and the amount present varies considerably in degree. There may be, as in this case, mainly "undifferentiated" tissue and the differentiation mainly recognizable is in the direction of bone marrow. In others there may be differentiation simultaneously in several directions, thus with the formation of several tissues.

The occurrence of haemopoietic tissue raises many points, mentioned earlier, about the relation of bone tumours to reticulo-

sarcoma and to tumours of the myeloma series but these will not be discussed at this stage. The essential feature that is emphasized here is the inter-relation of the connective tissues and haemopoietic tissue as applied to these neoplasms; thus it should be appreciated that bone marrow tissue is to be found in bone tumours in the same way as are cartilage, mucoid tissue, fibrous tissue and others.

As mentioned earlier the physical features of bone play an important part in influencing our ideas of the relationships of the tissues so it is important to recognize the kinship of the "hard and soft" tissues.

#### SUMMARY.

A case of osteolytic bone sarcoma, in which recognizable haemopoietic elements occur as an integral part of the growth, is described.

The essential inter-relationship of the various connective tissues, including haemopoietic tissue, is emphasized.

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## MALIGNANT GIANT CELL TUMOUR OF BONE.\*

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*Like — but oh! how different*

*Wordsworth; The Mountain Echo.*

CONFUSION has existed regarding several of the bone tumours but, when this state is examined, it is found to be due usually to a too rigid conception of the nature of the growths. It is usual for us in pathology and surgery to make compartments—often with the best of intentions and with good reason. Next, however, we attempt to mould or otherwise deform the prospective occupants to make them fit, and then we feel offended because ultimately they demonstrate a resilience that prevents permanent conformation with our preconceived pigeon-hole. This applies particularly to the giant cell tumour.

Many tumours contain giant cells either of the tumour or the foreign body type (or both) and the term Giant Cell Tumour could be (and sometimes has been) applied to any of them. Here the term is reserved for those in which there is a large number of foreign body giant cells of the osteoclast type.

### BENIGN GIANT CELL TUMOUR.

The benign giant cell tumour is a characteristic growth; the special features which distinguish it from other bone tumours are its frequent localization to the growing ends of the long bones, its sharp demarcation from the adjacent shaft, its expansion of the bone, its failure to extend beyond the confines of the bone and the characteristic radiological appearance (trabeculation).

Histologically it is just as distinctive. There is a pleomorphic stroma composed of spindle cells, some small round cells and other wandering cells in various proportions in different cases, but the outstanding feature is the large number of multinucleated giant cells of the osteoclast (that is, foreign body type—Fig. I).

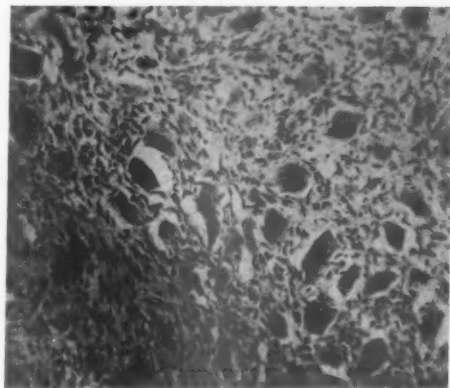


FIG. I. Photomicrograph of a section of a giant cell tumour showing the characteristic giant cells. Most of the stroma is of the pleomorphic type but the lower left corner shows a spindle cell tissue. (x 130.)

These macroscopic and microscopic features have led to its being regarded as a tumour distinct and clearly defined from other bone growths. A considerable amount of discussion has ranged round the point as to whether it is actually a neoplasm or an inflammatory condition.

It might be said at the outset that we are not dealing with one condition. Thus any widely embracing statement can be shown to be wrong in some or other case. Even if we were to regard the condition as a unit, then it is essential that we should understand that it has an ill-defined periphery and merges into other conditions.

In 1922, Stewart put forward the idea that the condition was a neoplasm and that the essential feature of the tumour was the giant cell—the osteoclast; hence the tumour

\*Read at the Annual General Meeting, Sydney, June, 1951.

has been designated an osteoclastoma. This is based on the assumption that the osteoclast is a specific cell capable of reproducing itself.

All the evidence, however, would indicate that the osteoclast is a foreign body giant cell, a particular response of any of the connective tissue cells of bone to a special stimulus. There is no suggestion that originally such a stimulus even remotely resembles a neoplastic one; indeed osteoclasts are ubiquitous and are found in almost all bone disturbances of whatever kind. It is difficult to see how cells responding in this manner and in what is clearly an unstable state could be fixed in this form and yet be able to reproduce themselves. There is no *a priori* support for such an idea and, what is more important, there is no support for it anywhere in biology. That the cells which produce the giant cells may become the cells of the tumour is, of course, beyond any question but that the giant cells themselves constitute a specific cell lacks substantiation. It might be said here, as a maxim, that where some new biological principle is invoked to explain a phenomenon, such explanation is almost certainly wrong; pathological changes are merely the result of normal biological phenomena (that is, cellular activity) different from the usual only because and in so far as the circumstances are different.

There are three main reasons for thinking that the essential cells of the tumour are not the giant cells.

1. The number and distribution of these cells vary considerably. The number of the cells ranges from a few scattered in a fibrous stroma to such numbers that they completely monopolize the microscopic field. As will be seen it is possible to change the number of these cells by interference with the tumour.
2. The cells are similar in kind in tumours of very different degrees of malignancy, that is, they are identical in benign and malignant tumours; but this does not apply to all the stromal cells.
3. When haemorrhage or infection occurs in lesions such as the fibrous areas or cysts of osteitis fibrosa associated with

parathyroid disease, many giant cells are to be found and indeed a condition indistinguishable from the benign giant cell tumour occurs.

That many of these tumours are not true neoplasms is indicated by several observations such as:

- i. their occurrence either as part of or associated with fibrous and cystic areas in osteitis fibrosa.
- ii. their response to simple surgical measures or to radiotherapy.
- iii. the gradation between well developed forms and those with no giant cells, such cases showing no neoplastic features.
- iv. tissue proliferations where stromal and giant cells appear in relation to zones of haemorrhage (Fig. II).

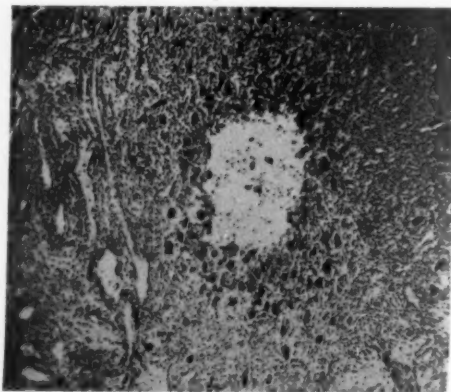


FIG. II. Photomicrograph of a section of a giant cell tumour showing giant cells accumulated especially round an area of degeneration and haemorrhage. (x 30.)

The principal argument in favour of the neoplastic nature of these conditions is that there are examples which show invasive features and metastases. It will be appreciated that this is due to a fixed idea that all conditions with a morphological resemblance — remarkably close in some cases — must of necessity be the same condition.

This notion has been the cause of much of the confusion. On the one hand there is more than one condition which presents the morphological, radiological and histological

appearances and on the other (as happens with all diseases) it is not possible to find a sharp dividing line between what usually, in their more characteristic forms, are easily distinguishable and separable.

We will now examine the form of the growth which resembles and yet differs from the benign giant cell tumour.

#### MALIGNANT GIANT CELL TUMOUR.

At first sight the malignant giant cell tumour closely resembles the benign form. The example which, to begin with, we may regard as typical arises at the ends of the long bones, is at first sharply delimited and has the characteristic (trabeculated) appearance on X-ray examinations. Its histological appearances are often at an early stage, so similar to those of the benign tumour that they are indistinguishable.

It differs from the benign tumour, however, in the recurrence after treatment to which the benign form responds, by invasion of the surrounding tissues (sometimes even before interference [King, 1933]) and later the development of metastases.

Some forms of this tumour presents features which enable it to be distinguished readily. It may arise in places other than the long bones: indeed the spinal vertebrae (either bodies or processes) are frequently affected. Tumours in unusual sites should be regarded from the outset as probably not benign types.

Those in which rapid growth and invasion through the cortex of the bone at an early stage occurs indicate gross malignancy. This is apparent macroscopically or radiologically, and histologically there is usually an anaplastic tissue which resembles that of an osteogenic sarcoma. It is apparent in these cases that the giant cells are incidental.

The extreme examples therefore present no special problem and difficulty arises only in those cases in which there is a close similarity of the benign and malignant form. The difficulty with these cases is accentuated by examples in which malignant change becomes apparent (and perhaps develops) in what appeared to be typical cases of benign giant cell tumours (Bogart, and Imler, 1947).

#### DIAGNOSIS OF BENIGN AND MALIGNANT TUMOURS.

The problem of distinguishing these two forms may occur in any part of the skeleton but is usually most serious in tumours occurring at the ends of the long bones.

As already mentioned the extreme form of each presents no difficulty. Not only does the typical benign tumour present a characteristic form but it does not show the progressive features of a true neoplasm. The occasional retrogression of one of these after fracture of the bone indicates its essentially non-neoplastic nature. The pleomorphism of the stromal tissue also supports this view. However, there are more persistent examples and these must be distinguished from the benign form.

Unless the condition is observed for a considerable time there is no certain gross morphological or radiological criterion of diagnosis. Resort is thus necessary to examination of biopsy material obtained either for this express purpose or during operation for attempted cure of the condition.

Examination of such material immediately shows that the giant cells—the osteoclasts, as might be anticipated, give no indication whatever as to the prognosis of the condition (Aegerter, 1947). They vary in number and form: they may be scarce or may predominate in the sections and the number of nuclei in a cell may be small or large.

The important distinguishing feature is the stroma (Jaffe *et alii*, 1940). The examples which, as shown by subsequent course, are benign have a pleomorphic stroma showing a mixture of various fixed and wandering cells. On the other hand the malignant tumours show the significant feature of neoplasia, namely a more uniform structure—that is to say, cells of close lineage. These may be largely spindle cells (Fig. III) though mixed spindle and irregular polyhedral types may be found.

One important feature of the benign tumour is that haemorrhage is common (and is responsible for its characteristic colour) and giant cells are usually more numerous in relation to areas of haemorrhage (Fig. II). It is apparent that such areas of haemorrhage may occur in the malignant

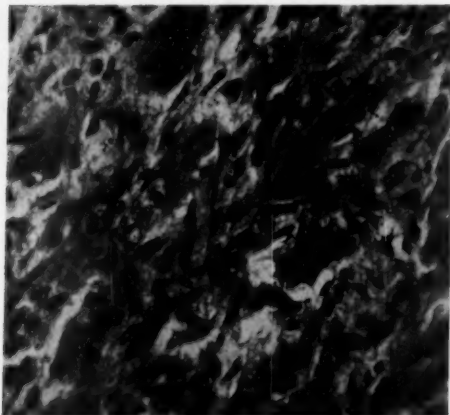


FIG. III. Photomicrograph of a section of a malignant form of giant cell tumour showing the spindle cell type of tissue. (x 275.)

tumour and this will add greatly to the difficulty of diagnosis. Indeed it is a matter of distinguishing areas of spindle cell tissue amongst the more pleomorphic stroma and attaching appropriate significance to this.

The difficulty is well exemplified by one case in which a tumour of the lower end of the tibia was diagnosed provisionally as a giant cell tumour of doubtful malignancy. A biopsy specimen was obtained at open operation. The section showed many giant cells, some pleomorphic tissue but some areas of spindle cells which suggested a malignant tumour (Fig. I). As there was doubt a further specimen was taken which histologically seemed indubitably benign because only giant cells and pleomorphic tissue were present. A diagnosis of benign tumour would have been given but enquiry revealed that a mild infection of the wound had occurred before the second specimen was taken and it was noted that old haemorrhage was present in the tissue. It was clear that any true neoplastic tissue was probably quite overshadowed by the inflammatory change following (operative) injury, haemorrhage and mild infection. Thus the diagnosis of a benign tumour in this second specimen would have been due to the misinterpretation of what was, from the point of view of the original condition, an artefact. This indeed was the case since the malignancy of the growth was soon clinically apparent.

A degree of uniformity of cell type in the tumour, within the wide limits which are found in neoplasms, is in uncomplicated cases easily distinguishable from the cellular diversity seen in inflammatory states either bacterial or chemical in origin. The rate of proliferation also is usually indicated by the discovery of mitotic figures (Fig. IV).

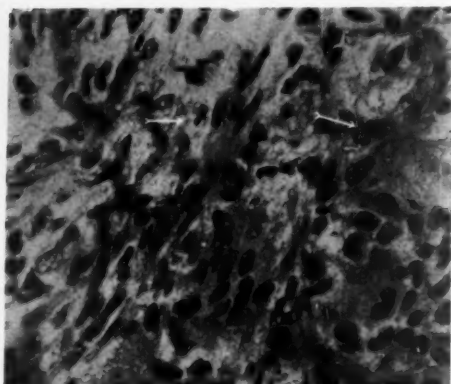


FIG. IV. Photomicrograph showing spindle cells between the giant cells and (indicated by arrows) nuclei undergoing mitosis. (x 275.)

The important things therefore in the histological diagnosis of the malignant tumour are the homogeneity of the tissue stroma (the osteoclasts being ignored) and the presence of mitotic figures. Any superimposed inflammatory changes which are much more significant here than tumours in other parts of the body (because less readily disentangled), must be carefully assessed.

Radiologically there is a similar difficulty in diagnosis. Features which will attract attention are irregularity of the trabeculation and invasion beyond the cortex. Here error may occur easily because when decalcification occurs the actual position of the periosteum is uncertain and a soft shadow may be beyond the normal extent of the bone, but if the cortex has been expanded and decalcified it may still not yet have invaded the soft tissues. The relation of a soft tissue shadow to actual decalcified bone is of paramount importance and should not be dismissed or determined on inadequate grounds.

It is this kind of difficulty that has been responsible for so much of what is a very real confusion. As has been shown a malignant tumour may be diagnosed, with all the misapplied confirmation of an histological opinion, as innocent whilst on the other hand extreme resorption of bone may lead to the opinion, morphological or radiological, that a benign condition is invasive.

We have emphasized the lowly malignant forms here but the malignant giant cell tumour has a range of malignancy which extends from a slow but progressive form of the benign giant cell tumour to a rapidly growing neoplasm which may be amongst the most malignant of the osteogenic sarcomata.

This tumour thus constitutes a link between the benign giant cell tumour (which in its truly benign form is probably not a neoplasm) and the osteogenic sarcoma and emphasizes the point that Nature does not have water-tight compartments, attractive and comforting though these may be to us.

#### SUMMARY.

1. The malignant giant cell tumour of bone is described here as the malignant growth which contains many foreign body giant cells (osteoclasts).
2. This tumour is at first localized to one part of a bone, radiologically shows

characteristic trabeculation and histologically shows a structure closely resembling that of the benign giant cell tumour.

3. The features distinguishing it from the benign giant cell tumour are the relatively uniform stroma and the presence of mitotic figures.
4. It is the less malignant form of this tumour, and the lack of clear segregation of it from the benign giant cell tumour that is responsible for much of the confusion regarding the benign tumour.
5. Difficulties of histological diagnosis are due to foreign body giant cells and a superadded pleomorphic stroma occurring in malignant tumours as the result of haemorrhage and infection.
6. The benign giant cell tumour does not fulfil the criterion of a neoplasm, namely that of progressive growth, and thus its distinction from the true neoplastic form is extremely important.

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# POSTERIOR DISLOCATION OF THE CERVICAL SPINE.

By J. T. HUESTON.

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*In cases where the vertebrae are curved inwards from a fall or the impact of some heavy weight, no single vertebra is much displaced from the others as a rule; but if there is great displacement of one or more, it brings death.*

(Hippocrates, ON JOINTS, XLVIII.)

IN this paper attention is drawn to the injury of the cervical spine resulting from hyperextension, namely a posterior dislocation of one vertebra on the next below. Rupture of the anterior longitudinal ligament is a feature of this dislocation, and quadriplegia is always present. Hence the recognition of such dislocations is of importance. No distinction is usually drawn between the incidence of quadriplegia in flexion and in extension injuries of the cervical spine. Its context makes it apparent that the quotation from Hippocrates refers to the hyperextension type of injury.

## INCIDENCE OF POSTERIOR DISLOCATION

In the five years ending June, 1950, there were 44 cases admitted to The Royal Melbourne Hospital with injury of the cervical spine.

After excluding injuries of the atlas and axis, 37 cases remain for consideration. This clinical division of the cervical vertebrae into the upper two, and lower five is justified both functionally and anatomically (Eastwood, 1940).

Of the 37 patients with injuries of the lower five cervical vertebrae, 9 showed simple crush fractures with no antero-posterior displacement and all survived. The remaining 28 cases showed some displacement and 12 of these died. Therefore some degree of dislocation at the level of injury was present in every fatal case.

Contrary to the usual teaching, this displacement did not always occur anteriorly. Twenty-five cases were of the flexion injury type, with fracture-dislocation and anterior displacement of some degree. However there

were 3 cases of hyperextension injury with posterior dislocation. This posterior dislocation was always associated with quadriplegia which was always fatal.

TABLE

	Number of Cases	Number of Deaths
<i>Flexion Injuries.</i>		
Simple Compression Fracture	9	—
Fracture-dislocation		
(a) with quadriplegia	6	5
(b) without quadriplegia	19	4
<i>Extension Injuries.</i>		
Posterior Dislocation	3	3
(all quadriplegic)		

## PATHOLOGY OF POSTERIOR DISLOCATION

### (a) Radiological examination.

In posterior dislocation there is displacement of one vertebra backwards on the next below it. The spinal cord is narrowed at this level by approximation of the inferior border of the dislocated vertebral body to the upper border of the lamina below. There is separation of the facets on the articular processes, which is very pronounced in Fig. 1. This feature contrasts with the overriding and interlocking of these processes seen in anterior dislocations.

There may be obvious angulation at the level of dislocation, so that the intervertebral joint space is widely opened anteriorly. This is shown in Fig. II, in which the small fragment separated from the inferior margin of the third cervical vertebra presumably was avulsed by the ligaments attached.



FIG. I. Lateral radiograph of the cervical spine of a patient with posterior dislocation of the sixth cervical vertebra on the seventh. Note wide separation of articular processes.



FIG. II. Lateral radiograph showing posterior dislocation of the fourth cervical vertebra on the fifth due to hyperextension.

#### (b) Autopsy examination.

Figs. III. and IV. show a block of vertebral bodies including the third to sixth cervical vertebrae. These are the vertebrae which are shown radiographically in Fig. II.

There is some dislocation posteriorly of the fourth cervical vertebra on the fifth. There is gross tearing of the anterior ligamentous structures, allowing the intervertebral joint space to be widely opened anteriorly. The inferior border of the dislocated vertebral body has bulged the posterior longitudinal ligament back into the spinal canal (Fig. IV). The spinal cord is severely compressed at the level of the dislocation. It is clear that originally this compression was between the protruding posterior border of the body of the fourth cervical vertebra anteriorly, and the upper border of the lamina of the fifth cervical vertebra posteriorly. There was no laceration of the cord, but the compression had reduced its thickness to 3 mm. Total quadriplegia was present. Death from respiratory failure occurred forty hours after the injury.

#### COMMENT.

All three cases of posterior dislocation presented the same clinical course of total quadriplegia rapidly followed by death from respiratory failure. However, autopsy was performed upon only one case. It is suggested that similar gross ligamentous and cord damage was present in the two other cases which presented similar clinical and radiological pictures.

The mechanism of production of quadriplegia in dislocation of the cervical spine is accepted as due to compression of the spinal cord, either between the displaced vertebral body and the laminae, or by protrusion of the intervertebral disc substance (Jefferson, 1940).

The much higher incidence of cord damage in this type of dislocation may be explained mechanically. During an anterior dislocation of one cervical vertebra on the next below it, there is an increasing resistance to displacement due to the inclination of the inter-articular joint surfaces. Once this has been overcome, and interlocking is



FIG. III. Anterior aspect of the cervical vertebrae shown in Fig. II showing gross tearing and separation of the anterior ligamentous bonds.

present, there is no resistance to further displacement and cord damage may result. In hyperextension injuries, however, once the anterior ligamentous bond between the vertebral bodies is ruptured, nothing remains to resist further posterior displacement. Separation of the articular processes is observed. This is the probable explanation of the severe degree of cord damage.

It is generally accepted that one can rely upon the integrity of the strong anterior longitudinal ligament in the management of spinal injuries. This is true in cases with flexion injury of the vertebral bodies, but does not apply to dislocation due to hyperextension. Where there is posterior dislocation and disruption of the anterior longitudinal ligament, skeletal traction in flexion or the neutral position is required. Extension is to be avoided at all costs, since this is the mechanism of injury.

The reduction is unstable, the vertebrae being without intact retaining ligaments. Hence frequent radiological confirmation of

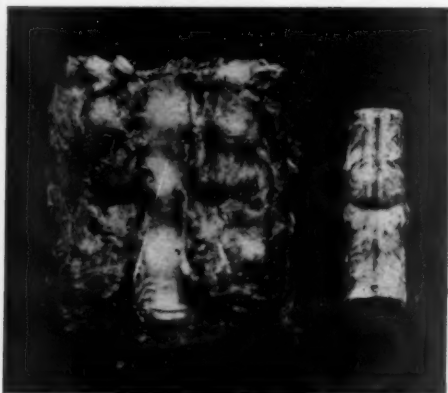


FIG. IV. The same specimen as shown in Fig. III after removal of laminae, showing anterior wall of the vertebral canal. The anterior wall is bulging back at the level of dislocation. The spinal cord shows the constriction present at this level.

reduction is necessary. Manipulation of these dislocations is unnecessary and may be dangerous.

The prognosis is grave in posterior dislocation because of the universal incidence of severe cord damage in those cases so far observed. However, reduction of the bony displacement should always be attempted so that any ischaemic element in the cord damage may be relieved.

#### SUMMARY.

1. Posterior dislocation of the cervical spine is described, and contrasted with the more common type of anterior dislocation.
2. The mechanism of injury is hyperextension, with disruption of the anterior longitudinal ligament.
3. In all cases quadriplegia was present. The anatomical basis of this is discussed.
4. The application of these findings to the management of cervical spine injuries is discussed.

#### ACKNOWLEDGEMENTS.

I wish to thank Mr. Eric Price for his helpful suggestions. The advice of Dr. R. A. Joske and F. J. Bromilow, M.Sc., arising out of many discussions has been invaluable.

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## CASE REPORT.

### INTERDIGITAL PILONIDAL SINUS CAUSED BY WOOL.

By A. D. MATHESON.

Hamilton.

**F**OUR cases of interdigital pilonidal sinus formation were reported by King (1949), occurring in barbers and due to hair penetrating the skin in the interdigital space. It would be expected that a similar condition should occur in others whose occupation is of a similar nature, and in this case an identical process developed in a man who was a shearer; wool fibre being the cause of the condition.

#### Case Report.

R.J.B., male, aged 34 years, a shearer for the past fifteen years, presented on the 6th March, 1951. Twelve years previously, he first noticed a painful swelling develop in the interdigital space between the left middle and ring fingers. After incision by his doctor, pus drained freely and healing occurred seven days later. He had no further symptoms until two years ago when, over a period of seven days, a similar type of swelling recurred in the same area. After incision, healing again took place after seven days. Three months ago he

noticed a further recurrence of the swelling, which developed more slowly than on the two previous occasions. He applied hot fomentants to the affected area and a spontaneous discharge of pus occurred, not in the web between the fingers where the previous incisions had been made, but nearby on the dorsum of the hand. Alternate healing and discharging of pus followed; later a sinus also developed in the web between the fingers. This condition continued until he was seen on 6th March, 1951.



FIG. I. Photograph of the left hand of the patient showing the sinus opening in the interdigital space between the ring and middle fingers.



FIG. II. Photomicrograph of a section of the superficial part of the sinus, cut slightly obliquely, showing the lining of squamous epithelium and some debris including a piece of wool in its cavity. (x 90.)

Six months previously, between the same fingers of the other hand, he experienced pain on movement of the ring and middle fingers and noticed redness of the skin in the interdigital space between these fingers. He treated the condition himself by immersing the hand in hot water each evening and with the index finger and thumb of the other hand, he squeezed the affected area. One evening during this procedure, a fibre became visible, projecting from the skin in the interdigital space. With the aid of household tweezers, he pulled out a fibre, he estimated to be about three-quarters of an inch in length. He stated that after close examination of the fibre, he was quite certain it was wool. The pain and redness then gradually disappeared and he has had no recurrence.

On examination of the left hand, a small sinus and scar were visible in the interdigital space between the middle and ring fingers. Another small scar was also seen nearby on the dorsum of the hand between the fingers. There was induration of the soft tissues in this area. No discharge was observed at the time of examination, and the inflammatory process appeared quiescent. At operation, a block dissection of the affected area was carried out. The interdigital blood vessels were dissected free and preserved. The cavity that remained was swabbed out with a detergent antiseptic and the skin approximated with interrupted sutures. Primary healing occurred and the patient resumed shearing one week later.

#### Pathological Report.

The specimen consists of an ovoid piece of skin in the middle of which there is a distinct sinus. Projecting from this sinus there is a small piece of material of uncertain nature (which is left *in situ* for section).

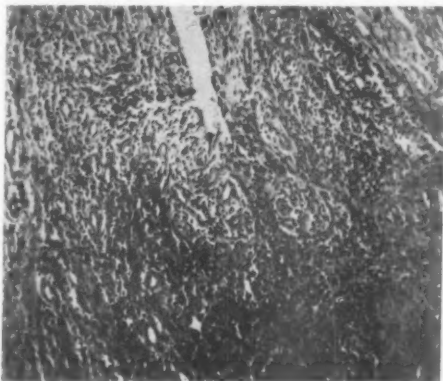


FIG. III. Photomicrograph of a section of the deeper tissue showing the cellular infiltration in the neighborhood of the sinus which is here not lined by an epithelium. (x 180.)

Histologically there is a sinus which, in the superficial part, is lined by squamous epithelium and, in the deeper portion, has a connective tissue surface without epithelium.

In the cavity there is debris included in which is a hair-like structure. This differs from human hair in having a somewhat

serrated margin and in showing a slightly (and irregularly) segmented appearance. These two features are characteristic of sheep's wool and are not found in human hair. In the tissue round the sinus there are numerous wandering cells, these extending for some distance into the surrounding tissue.

The presence of the sheep wool in the sinus is a further demonstration of the extrinsic origin of pilonidal sinuses, particularly in the interdigital space.

#### DISCUSSION.

Concerning the mechanism of penetration of a wool or hair fibre through the skin in the interdigital space, it appears reasonable to postulate that once the end of a fibre has entered a small depression or cleft in the skin, a particular type of movement by adjacent two fingers in full adduction, may act as a propelling force to the fibre.

The aim of treatment of these sinuses is the same as for those occurring in the sacrococcygeal area, namely excision of the sinus or sinuses together with the surrounding indurated tissue, approximation of the remaining tissue edges and healing by first intention. If the sinus is temporarily closed and the patient presents with an abscess, adequate incision to obtain free drainage, plus chemotherapy will allow the inflammatory process to become quiescent. While such a condition exists, healing by first intention can usually be achieved after a block excision of the affected area; avoiding opening into the sinus and using further chemotherapy.

#### SUMMARY.

1. A case of interdigital pilonidal sinus is reported.
2. The etiology and management of this condition is discussed.

#### REFERENCE.

- KING, E. S. J. (1949), *Aust. N.Z.J. Surg.*, vol. 19, page 29.

## Books Reviewed.

### CLEFT PALATE AND SPEECH.

By MURIEL E. MORLEY, B.Sc., F.C.S.T. Second Edition. Edinburgh: E. & S. Livingstone Ltd., 1951. 4½" x 7½", xx plus 160 pp., 56 figures. Price: 12s. 6d. net.

The earlier edition of this excellent book has been recommended as a standard work on all aspects of the subject by the College of Speech Therapists, London.

It is a work of great value to students, for facts are so well clarified regarding cleft palate surgery (advocating a method that produces an excellent functional result as well as a sound anatomical one), the analysis of speech defects associated with cleft palate speech and suggestions for treatment.

The necessity for co-operation between the surgeon, the orthodontist and the speech therapist working as a team is advisedly stressed in the Foreword by Professor T. Pomfret Kilner, C.B.E., F.R.C.S.

Such improvements as Miss Morley has introduced into this second edition have undoubtedly enhanced the established value of her book.

### THE RHESUS DANGER.

By R. N. C. McCURDY, M.B., Ch.B., D.P.H. London: William Heinemann Medical Books, 1950. 4½" x 7½", 138 pp. Price: 5s.

Dr. McCurdy has, from personal experience, had cause to ponder over the problem of the Rhesus danger. Two of his own children have died from haemolytic disease of the new born, so with this background he has written this book on the Rhesus danger, its medical, moral and legal aspects. The book is written simply and clearly for a lay reader. In part one he deals with the medical background of the problem. In excellent sequence he explains from first principles the various human blood groups and the story of the discovery of the Rhesus factor. The origin of haemolytic disease of the new born is then explained. The treatment of the disease is discussed at length. The use of premature induction of labour by artificial rupture of the membranes has been abandoned in many clinics because the results have not been as good as in those cases allowed to deliver themselves at term. Part two deals with wider issues. The author discusses at length and from all angles various solutions to the problems arising from Rhesus incompatibility. The possible solutions discussed are contraception, sterilization, abortion, artificial insemination and divorce. As he says "there can be no final opinion. There can be and should be opinions but no final ones." One fact that does not receive enough emphasis is that of the Rh negative (female) Rh positive (male) marriages only 4% ever have children who suffer from haemolytic disease of the new born. With this fact in mind the author does not discuss the problem as to whether the obstetrician should tell the expectant mother that her blood is Rh negative. This is a thoughtful book supplemented by a full bibliography and a good index.

### ATLAS OF HUMAN ANATOMY, DESCRIPTIVE AND REGIONAL.

By M. W. WOERDEMAN, M.D., F.R.N.A.Sc. Volume 2. London: Butterworth & Co (Publishers) Ltd., and Amsterdam: Wetenschappelijke Uitgeverij, 1950. 6½" x 10", x pp., 641 plates, plus index. Price: 82s.

The second volume of Professor M. W. Woerdeman's "Atlas of Human Anatomy" devoted to splanchnology, angiology, nervous system and organs of sense has now been published. This part of the Atlas does not maintain the high standard set by the first volume although some of the sections, notably the nervous system, are excellent.

The section on the lymphatic system could be improved, particularly the representation of the aortic chain of glands, the various groups of which are hardly more than indicated; it is also felt that the lymph drainage of the uterus and vagina, as represented, is misleading.

The section on the lungs would be improved by the inclusion of illustrations of casts of the bronchial tree. It is also suggested that diagrams showing the relations of the major viscera would improve the Atlas from the student's point of view.

In spite of these minor criticisms the work as a whole is excellent. Professor Woerdeman has produced an Atlas which will prove of considerable value to both student and post-graduate alike.

### JOHN HUNTER.

By S. ROODHOUSE GLOYNE, M.D. Edinburgh: E. & S. Livingstone Ltd., 1950. 9" x 6½", x plus 104 pp., portrait and 16 illustrations. Price: 15s.

In this volume the author gives us a good account of the life and times of John Hunter. It is well written and documented and gives an adequate picture of Hunter's enormous variety of research, his relations with his brother and his friendship with some of his contemporaries notably Edward Jenner. To those who want to know a little more about John Hunter, this book can be recommended.

### ARCHIVES OF THE MIDDLESEX HOSPITAL.

Edited by DOUGLAS McALPINE. Volume 1, Number 1, January, 1951. Published quarterly. Edinburgh: E. & S. Livingstone Ltd. Subscription, £2 2s. per annum.

This Journal, the first number of which now appears, is a resurrection of that of the same name which appeared earlier in the century. It was begun in 1902 as laboratory reports and expanded to include clinical material in 1905. Publications ceased during the Great War in 1916.

This publication is of very high standard, it contains articles on a wide range of subjects, written by prominent clinicians. The material is of great interest and the presentation of it is extremely good. The text is clearly set out and the illustrations are particularly well done.

The appearance of new journals at a time when so many are being printed is to be deprecated but it must be granted that the Middlesex Hospital has, in view of the old Archives, some prior rights in the field of medical publications. There can be no

doubt that if the standard of this first number is maintained, the Journal will be a useful and justifiable addition to medical literature.

**"CHIRURGIE RÉPARATRICE ET CORRECTRICE DES TEGUMENTS ET DES FORMES."**

By L. DU FOURMENTELLE. 2nd Edition. Paris: Masson et Cie, 1950. 6½" x 9½", 400 pp., 457 figures. Price: 1650 fr.

This book represents a useful contribution to the literature on reconstructive surgery which is still relatively scanty when compared with the vast amount of writing which has been completed by other branches of surgery. Its author's work has been well known for very many years and, if for no other reason than this, the book is worthy of the attention and study of all those concerned in the practice of plastic and reconstructive surgery. Plastic surgeons, trained in the British schools, while appreciating the wealth of experience that has gone into its making, will perhaps regret the emphasis given to the cosmetic side of the work at the expense of that vast and infinitely more important field which concerns restoration of function. Reparative and corrective surgery in the hands of most plastic surgeons to-day extends over a very much wider field with a very much wider choice of procedures than indicated here.

The book opens with the section on general principles and technical finesse in regard to suturing which sets a standard which can hardly be said to be upheld in many of the cases illustrated throughout the book. It is somewhat of a shock, for example, to read in the text of the infinite care with which wound suturing is carried out, and then to be faced with such illustrations as Figs. 94, 103-104 and 416.

In the section on rhinoplasty the important question of lining receives scant attention and indeed, in such illustrations as Figs. 250-251, and 256-257, appears to have been completely disregarded. There are many other features such as lining the mouth with hair-bearing skin (Figs 348, 349) with which other surgeons would disagree strongly, and perhaps the only section that can be regarded as in any way complete, is that on the corrective surgery of the nose.

A few publication defects such as the inclusion of poor quality photographs, a confused transposition of diagrams (Figs. 279, 280), and some others could be corrected with advantage.

The value of the book, however, lies in the fact that it is a record of the experiences of one who has practised the specialty for very many years and it will give many in other countries an opportunity to compare continental methods with their own.

**PRINCIPLES OF PATHOLOGY.**

By Professor R. A. WILLIS. London: Butterworth & Co. Ltd., 1950. 6½" x 9½", xi plus 722 pp., 288 illustrations. Price: 50s.

A new book on pathology by Professor Willis is an event of some importance, as his previous publications on the spread of tumours in the human body and on the pathology of tumours very quickly achieved a considerable reputation. This present volume is of a different character. It is aimed at the undergraduate, and attempts to cover the whole field of pathology as far as the undergraduate

student may be expected to grasp it. This is a formidable task for a single author, and it is made still more difficult in view of the fact that academic pathology, certainly one of the most conservative of the medical sciences, is now showing signs of being influenced by the results of the great advances of the last 30 years in cytology, cellular biochemistry and the application of physiological and experimental techniques to pathological problems. There is now in consequence a good deal more discussion than usual on how much, if any, of this more functional and biochemical approach to the subject ought to be incorporated into undergraduate courses in pathology, which are still solidly (and rightly) based on morbid anatomy. Again the decision has become still more difficult on where the balance should be struck in a text designed for undergraduates between on the one hand an avoidance of anything uncertain or controversial, with the expression of definite opinions which students can memorise—in short, dogmatism; and on the other a record of a bewildering and often conflicting mass of experimental opinions out of which nothing firm or readily remembered may have emerged. The problem is perhaps a little more easily tackled in the post-graduate field, as for example, by the present insistence of the surgical Colleges on a much more fundamental approach than hitherto to general pathology as a proper foundation for later studies in surgical pathology.

Therefore one opens a new text book dealing with the whole field of pathology with great interest and still more so in the present case as Professor Willis treats the subject as a whole, without the customary division into "general" and "special" pathology. This has the advantage of bringing together matters which in the usual systematic text book are separated, and of avoiding some repetition. It is not, however, an unmixed blessing, as it has in other cases the disadvantage of splitting up topics which usually fall together. For example, "cirrhosis of the liver" is here split into a dozen different facets as also are the various aspects of congestive cardiac failure.

Such an approach naturally invites comparison with the celebrated text book of W. G. MacCallum who also attempted, a generation ago, a synthesis of the subject on rather similar lines, and whose excellencies of description and style earned him a large measure of success. The comparison is, in fact, a very interesting one as Professor Willis writes with the vigour, clarity and courage of conviction which those who know and admire his other books have come to expect. It is, however, a little disappointing, as the present volume, with the exception of the section on tumours, is rather of the nature of an amplification of lecturer's notes, so that the subject is split up into a very large number of quite short sections each with its headings, subheadings and tabulations. Only essentials are given. Pneumonias, for example, are allotted less than 7 pages; fever less than eleven lines.

As teachers we preach what we practice, and Professor Willis therefore adopts a conservative and mainly morphological approach to his subject. The more functional and biochemical aspects of pathology are touched on only very lightly. After an introductory chapter the work begins with a

succinct account of uncomplicated repair, as manifested in tissues of different types, and of the formation of granulation tissue. Then follows a general account of acute and chronic inflammation. In this more emphasis might have been placed on the effects of the products of tissue injury and breakdown, about which some chemical knowledge has now accumulated. Again, not all will agree with the derivation of the plasma cell from the lymphocyte or with the transformation of lymphocytes into monocytes.

Six chapters are then devoted to inflammation and its consequences in particular sites, as illustrations of the application and modification of the fundamental inflammatory reaction in accordance with local factors. A short and quite elementary account of the morphology and classification of bacteria precedes a consideration of the diseases caused by particular organisms, but is amplified later by a useful chapter on immunity. In the latter the cellular aspects of antibody formation are not much emphasised, even allowing for the controversial nature of some of the evidence bearing on this field.

The chapters on tuberculosis and syphilis are noteworthy and should provide students with a most useful "birds-eye view" of these diseases. Those on diseases caused by fungi and by metazoal and protozoal parasites are rather more detailed than many others. A very useful and very complete chapter is entirely devoted to the reactions evoked by foreign bodies of all types, in which the unity in diversity of this reaction is very well brought out.

Congestive cardiac failure, as such, receives only brief mention. The time has surely come when teachers of pathology must concern themselves with the functional pathology of this syndrome, of which students see so much and about which they usually know so little, to a large extent because they still tend to think of it too much in morphological terms. To a lesser extent the same may be said of the pathology of anaemias. There might well have been included some mention of the important circulatory and respiratory adjustments which are common to all severe anaemias.

The reader naturally turns to the section on tumours with most interest for here Professor Willis speaks with real authority. It is rather longer than is usually the case in a book covering the whole subject. This, however, is no disadvantage. On the contrary it is a lucid and condensed summary of the author's large book on tumours, containing in about one fifth of the space the essentials of the morbid anatomy, histogenesis and behaviour of tumours, together with a short chapter on carcinogenesis. This should prove most useful to the undergraduate and postgraduate student. Candidates preparing for the primary fellowship may need to amplify the chapter on carcinogenesis by consulting some of the references given at the end of this chapter.

A final very good chapter deals with antenatal pathology, malformations and teratology, and there are three appendices. Of the latter the first is a list of Greek and Latin names and their meaning, after the style of that published by Charles Powell White some years ago. Another gives notes of some great pathologists, and in the third the student is given most useful advice on the examination and description of pathological tissues, both gross and microscopic, with suggestions on how to use the evidence so obtained to arrive at a diagnosis. It is the lack of just such a trained approach to a slide or specimen as is here advocated which handicaps so many undergraduate students of morbid anatomy.

There are 300 illustrations, many of them diagrams. Some of the latter are perhaps oversimplified. For example, Fig. 94 of an Aschoff node gives no real idea of the morphology of this lesion. The photographic reproductions are of high quality, particularly those of syphilitic bony lesions from specimens in the Hunterian museum. The coloured plate of leukaemic blood (plate 10) is not up to the standard of modern illustrations of blood diseases. At the end of each chapter is a short and very well chosen list of references, which the author intends should be consulted. Many of the references have a note appended setting out the particular features for which they have been cited. This is a very useful practice which one wishes more authors would follow.

The book is printed on good paper and is well produced. Few misprints were noted, but a word, apparently "simile," has been jumbled in the legend to Figure 91 and "inoculate" is misspelled on page 122.

Surgeons will find this book useful for its succinct and authoritative discussion on tumours. There is, however, not enough detail in the treatment of the rest of the subject, nor enough discussion of its more functional and controversial aspects to meet the requirements of postgraduate students, or even the better undergraduates, unless they have access to a good library and are able to consult the references cited.

## Books Received.

### CONFRONTATIONS RADIO-ANATOMO-CLINIQUES.

By M. CHIRAY, R. A. GUTMANN, J. SENEQUE.  
Fourth Edition. France: G. Doin et Cie, Masson et Cie, 1951. 13" x 10", 68 pp., 127 illustrations.  
Price: 1250 francs.

### L'ANESTHÉSIE FACILITÉE PAR LES SYNERGIES MÉDICATIONNEUSES.

By H. LABORIT. France: Masson et Cie, 1951 9" x 6½", 120 pp., 7 illustrations. Price: 500 francs

### HANDBOOK OF MEDICAL MANAGEMENT.

By MILTON CHATTON, A.B., M.D., SHELDON MARGEN, A.B., M.D., HENRY D. BRAINERD, A.B., M.D. Second Edition. California, U.S.A.: University Medical Publishers, 1951. 7" x 4", 508 pp. Price: \$3.00.

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